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TABLE OF CONTENTS

Classical Gout FREDERIC WOOD JONES, F.R.C.S., F.R.S. 561

Significance of Haemoglobin Level in Puerperium R. F. A. DEAN, M.R.C.S. 567

Research into the Origins of Delinquent Behaviour J. BOWLBY, M.D. 570

Speech Disorders in Childhood
MURIEL MORLEY, F.C.S.T., DONALD COURT, M.D., and HENRY MILLER, M.D. 574

Acute Laryngo-tracheo-bronchitis JOHN Z. GARSON, M.R.C.S., D.C.H. 578

X-ray Examination in "Pneumonia" N. WYNN-WILLIAMS, M.B. 579

Adrenal Cortical Tumours GEORGE LUMB, M.D. 582

Second Example of the "Duffy" Antibody
ELIZABETH W. IKIN, B.SC., A. E. MOURANT, D.M., and GERTRUDE PLAUT, M.D. 584

MEDICAL MEMORANDA

Recovery from Strangulated Obturator Hernia. M. C. WATERFALL, F.R.C.S. ..585
Tuberculous Meningitis During Pregnancy, Streptomycin Treated. F. F. KANE, M.D., M.R.C.P.I., D.P.H. 585
Examination of Young Children with Acute Appendicitis. ROBERT STRANG, CH.M., F.R.C.S. 586

REFRESHER COURSE

Acute Abdominal Disease. V. ZACHARY COPE, M.D., M.S., F.R.C.S. 596

GENERAL ARTICLES AND NEWS

The General Practitioner and the Maternity Service. D. W. JAMES, M.B., B.S., M.R.C.O.G. 598
World Food Problems 602
NOVA ET VETERA 604
Simple Gastric Suction Apparatus. M. D. SHEPPARD, F.R.C.S. 604
MEDICAL NOTES IN PARLIAMENT 614
MEDICO-LEGAL 614
UNIVERSITIES AND COLLEGES 614
EPIDEMIOLOGY SECTION 615
MEDICAL NEWS 616
BIRTHS, MARRIAGES, AND DEATHS 618
QUESTIONS AND COMMENTS 619

OBITUARY

J. A. Ryle M.D., F.R.C.P. 611
Bernard E. G. Bailey, M.R.C.S. 613
R. H. Wagner, M.D. 613
Robert d'Ernst, M.D. 613
Lilias Frazer-Nash, L.R.C.P.Ed. 613
Harold Sheldon, M.D. 613
L. G. Bourdillon, D.S.O., M.C., M.B. 613

LEADING ARTICLES

Medical Aspects of Crime 590
New Concepts of Gout 591
A New Blood Group 592
Fee for Sight Tests 593

ANNOTATIONS

Speech in Childhood 594
Bone as Structural Material 594
Terminal Cardiograms 595
Perforation of the Oesophagus 595

REVIEWS

Towards Diagnostic Precision 587
Physiology and Anaesthesia 587
Field Epidemiology 588
The Life Table 588
Fundamentals of Chemistry 589

SUPPLEMENT

Health Service.—OPHTHALMIC MEDICAL PRACTITIONERS—CIVIL SERVICE MEDICAL OFFICERS—INVENTORIES OF HOSPITAL EQUIPMENT — TEMPORARY RELEASES FROM H.M. FORCES—THREAT TO WITHHOLD SERVICES—LONDON N.H.S. PAYMENTS—REGISTRATION OF OPTICIANS—DEPUTIES AND ASSISTANTS—DAY AND NIGHT HOURS—HOSPITAL CATERING—SPECIAL INDUCEMENT FUND 75
A Visit to the Transatlantic Branches. E. GREY TURNER, M.C., M.A., M.R.C.S., L.R.C.P. 77
PRIVATE PRACTICE COMMITTEE 79
CORRESPONDENCE 80
ASSOCIATION NOTICES 82

CORRESPONDENCE

Maternity and the N.H.S. KEITH VARTAN, F.R.C.S.; CLAUDE TANGYE, M.D.; W. A. BELLAMY, M.R.C.S.; M. CHALK, M.R.C.S., and E. JOZEF, M.B. ..605
Psychology of the Surgical Patient. MALCOLM DONALDSON, F.R.C.S. 606
Ogilvie's Syndrome. A. M. ABRAHAMS, F.R.C.S. 606
Experiences with "Dipercol." F. MURRAY, M.B. 606
Red Blood Cell Diameters. J. A. BOYCOTT, D.M. 606
Antibiotics and Hydrogen Peroxide. A. KRAUS, M.D. 606
Tetanus Treated by "Myanesin." P. P. FOX, M.B., and A. H. BANTON, M.D. ..607
Q Fever in England. M. D. WARREN, M.B. 607
The Acute Throat. E. J. FAIRLIE, M.R.C.S. 607
Erysipeloid. C. J. Luke, M.B. 608
The Wrong Solution. A. S. PLAYFAIR, M.R.C.S. 608
Aerosol Treatment with Penicillin. E. H. JEANES, M.B. 608
Fatal Air Embolism in Artificial Pneumoperitoneum. V. G. RADCLYFFE, M.B. 608
Anaesthesia for Fenestration Operation. M. SWERDLOW, M.B.; S. A. MASON, M.B.; REX BINNING, M.R.C.S. 608
Insufficient Anaesthesia. VIOLET RUSSELL, M.D.; S. F. DURRANS, M.R.C.S.; CHARLES WELLS, F.R.C.S. 609
Suture of Ruptured Kidney. R. D. N. BISSET, F.R.C.S. 610
Atomic Weapons and the Medical Profession. W. V. CRUDEN, M.B. 610
Students' Style. W. HARKINS, M.B. 611

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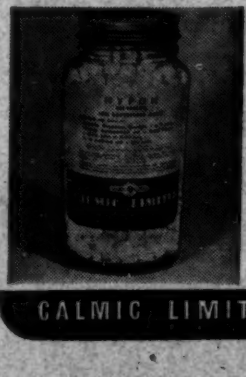

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY MARCH 11 1950

CLASSICAL GOUT*

BY

FREDERIC WOOD JONES, D.Sc., F.R.C.S., F.R.S.

"The Gout is so common a disease, that there is scarcely a man in the world, whether he has had it or not, but thinks he knows perfectly what it is."—William Cadogan in *A Dissertation on the Gout*, 1771.

It would seem that custom has sanctioned, and indeed almost imposed, a very definite form of introduction to an address such as this. To this accepted formula I am prepared to subscribe in so far as the usual opening consists in a general confession on the part of the speaker of his unworthiness to be accorded the honour of discussing the subject before so distinguished a body of experts. But I have noticed that there is commonly some passage made from this opening note of humility to a later and slowly emerging atmosphere of omniscience. This transition I shall not make. To the end I shall remain painfully conscious of the fact that upon the very partial knowledge of only a single example I am venturing to generalize upon a clinical condition of the utmost complexity.

We do not greatly value the opinions of a man who, having persuaded some woman to become his wife, assumes that he is the fount of all knowledge concerning the ways of women. We are not in the habit of attaching much importance to the views of any man who, on the strength of possessing a David Cox, poses as a great authority on British water-colours. There is far less reason why regard should be had for the ideas of one who, happening to own a little private gout, assumes that the secrets of that baffling disease are in his special keeping.

As an anatomist I shall naturally be excused from even pretending to any knowledge of the modern developments in the chemistry of the purine bodies; and I shall be pardoned, on the ground of ignorance, from making appeal to the latest dicta concerning the part played by uric acid in human metabolism. I shall therefore content myself by accepting the definition, which I believe to reflect present-day orthodox opinion, that gout "consists in a disturbance of purine metabolism marked by excess of uric acid in the blood." Unfortunately, there does not seem to be complete agreement among authorities on gout that even this definition expresses absolute truths. There are certain other pathological conditions in which hyperuricaemia is a constant feature and yet no signs or symptoms of gout are ever manifested (Cohen, 1948; Mason, 1949). On the other hand, it seems that all that may be said of the association of hyperuricaemia with gout is that "hyperuricaemia is *almost constantly* found" (Cohen) or hyperuricaemia *almost invariably* occurs at some stage in the course of the disease" (Mason). On general principles, it would seem logical to suppose that, though hyperuricaemia is a very common incident in the course of an attack of gout, it can hardly be regarded as the actual cause of it. Indeed, this appears to be the standpoint of the author of the latest orthodox pronouncement with which I am acquainted. Mason (1949) concludes that "the old saying that gout is a metabolic disorder, repeated in every textbook, is perhaps unfortunate. The more one studies the uric acid problem, the less do such abnormalities as exist appear to be fundamental." We are therefore left with our definition of the disease somewhat the worse for wear; but this is not to say that because it apparently does not express the whole truth it is not a perfectly good definition so far as it goes.

It must be made quite clear at the outset that the only condition I shall attempt to discuss is classical, or regular, gout. This clinical entity I shall not even designate by its accepted name as "articular gout" or "arthritic gout"; for, taking typical podagra as the classical manifestation of the disease, it would seem that it is not necessarily an arthritis, nor even an arthritic condition, in its primary manifestations, but is always and essentially periarticular in its situation. I shall also avoid the adjectival use of the term gout; for a perusal of the literature has persuaded me that the increasing loss of clarity in our conceptions of the essential nature of "periarticular gout" has been due in great measure to the common employment of the term "gouty" for all sorts of conditions that probably have nothing whatever to do with true classical gout. Having thus restricted the field to the discussion of a single but vividly individual entity, I am forced to confine myself, by reason of my lack of clinical experience, to what may be termed the natural history of a complaint the proper understanding of which demands so much more in the way of technical knowledge for its ultimate clarification.

Modern zoologists are at times guilty of discarding as "anecdotal" the observations made by the great naturalists of the seventeenth and eighteenth centuries. But it is to the credit of the physician that he has had more respect for tradition and has never neglected, nor despised, the work of the great writers on the natural history of disease. There is therefore no need to apologize for limiting my remarks to matters of simple observation of overt stages that appear to constitute the life history of a typical attack of periarticular gout.

Precipitating Causes

Whatever may be the underlying disturbance that is responsible for gout there is still needed some factor that fires the train of events witnessed in the progress of an attack. It is perhaps a gain to medical science, and to me it is a great personal satisfaction, that there is a tendency towards rather more charity being extended to the sufferer

*The Heberden Oration delivered to the Heberden Society on December 9, 1949.

by the physician. To-day there is not the universal condemnation—that "most people who have had the gout severely have deserved it"—which prevailed in John Hunter's time. We hear less of gluttony and of drinking to excess as being the inevitable precursors of an attack, and more charitable views include trauma, fatigue, stress, and allergic and psychic factors among the precipitating causes of an attack.

Whatever it is that pulls the trigger, there is no doubt that the attack, and especially the first attack, comes on with dramatic suddenness. In the poor small hours of the morning the patient is awakened on the sudden and at once passes from a condition of normal ease to one of acute disease by a transition which knows no light or shade, nor any stages of gradation. He is awakened by an intense and distinctive pain in his big toe.

Doubtless long familiarity with the condition leads to some forewarning by which the patient learns to recognize under what circumstances an attack is likely to occur. Sydenham said that in his case the "only forerunner was indigestion and crudity of the stomach"; but that, I think, must be regarded as a purely personal matter. It is probably a case of every man to his own choice in what he considers to be a warning of an attack. The overt objective signs of the attack are swelling and redness in the neighbourhood of the metatarso-phalangeal joint of the big toe. Following Morgagni (*De sedibus et causis morborum*), it is logical to make some attempt to ascertain the actual anatomical basis of the swelling and then to determine its cause.

Seat of the Disease

What is the actual seat of the disease? Does gout have its seat primarily in the joint and is it rightly termed an articular disease or an arthritis? All the older physicians apparently regarded the seat of the disease as being *in* the joint. Sydenham constantly alludes to the "affected joint," and such is the habit of most of the early writers on gout. Even to-day it is customary for British physicians to term the condition "acute gouty arthritis" (Cohen, 1948). From the supposition that podagra is an affection of the metatarso-phalangeal joint of the big toe arises all that literature in which it is accepted that gout is primarily an arthritic condition. It is here that gout, in British medical science, loses its identity, since it has become only one of many forms of arthritis; and gouty arthritis and rheumatic gout have become commonplace and accepted terms in medical literature.

That a typical attack of podagra is manifest in the neighbourhood of the metatarso-phalangeal joint of the big toe is patently true; but to postulate that the seat of the disease is *in* the metatarso-phalangeal articulation is a mere supposition: and this supposition would seem to have little real evidence to support it. Even Cohen, who consistently alludes to gout as an "acute arthritis," admits that "radiological changes [in the joint] occur late in the disease and are non-specific." Williamson (1920) declared, "in nearly two-thirds of all cases submitted [to *x*-ray examination] it not only failed to assist in the diagnosis but tended if anything to lead one to arrive at wrong conclusions"; and Mason (1949) states that "radiological changes which are present in about one-third of all cases" are of little diagnostic value. More than that, it is an incontrovertible fact that after repeated attacks of podagra extended over a period of more than thirty years there may be no signs whatever of arthritis or of any pathological changes in the joint revealed by *x*-ray examination (see Figs. 1 and 2). The dictum of Jaques Sédillot (1935) that "la goutte n'est pas une affaire articulaire" appears to be well justified.

FIG. 1.—The metatarso-phalangeal joint of the big toe of a man aged 71 who has suffered from podagra for 37 years. Taken at the termination of the acute stage of the attack.

FIG. 2.—The metatarso-phalangeal joint of the big toe of a man aged 56 who has suffered from podagra for 10 years. Taken at the termination of the acute stage of an attack.

The Swelling

The swelling, even early in the attack, occupies a wider area than that immediately overlying the metatarso-phalangeal joint. Commonly the big toe is involved right to its nail-bed, and the dorsum and medial side of the foot share in the swelling for some distance proximal to the joint. The skin is tense and shining, brilliantly red in the early stages of the attack, and the whole foot is notably dry and hot. It is natural to ask what it is that causes the swelling. Apparently all the early writers on gout considered that the swelling of the painful big toe was due to the accumulation of pathological exudates or humours in and around the joint.

Sydenham regarded it as "gouty matter" or "peccant matter." Oliver, of Bath, was more vivid in his description. "He represents the gouty matter as becoming acrid, tearing the vessels to pieces, curdling the synovia, and turning it into chalk" (quoted by Ring). All were agreed that some very deleterious humour was concentrated in the metatarso-phalangeal joint and that therefore the rest of the body was well rid of it. From such an argument there arose a belief that an attack of gout was a salutary disease, since the "peccant matter" had become separated and localized and one knew in which part of the body it had become distilled and isolated.

Passing for a moment the views of Caverhill (1769), the observations of John Ring (1811) are of considerable importance. He says: "It is the general opinion of ancient and modern physicians that gout proceeds from an accumulation of humours. Some thinking them a morbid secretion, and others considering them to be mere blood. The latter opinion appears more probable, *from the sudden manner in which the complaint comes on*." This is, I believe, the first expression of the fact that such a dramatically sudden formation of a tense red swelling is more likely to be caused by a sudden change in vascularity than by the accumulation of exuded humours of any kind. But Ring was not a pioneer in supposing that the swelling might be due to distended blood vessels.

Caverhill, in 1769, had given a clear and coherent expression of a highly confused theory which postulated that the

cause of the swelling, and of the pain, in podagra was due to a circulatory upset in the metatarso-phalangeal region of the big toe. Sydenham (1683) had already called attention to the fact that, in his own case, in the early stages of the attack, "only a remarkable swelling of the veins appeared as is usual in all gouty fits." Mason (1949) has called attention to the fact that Garrod (1876) depicted a big toe, in an acute attack of gout, with engorged vessels around the metatarso-phalangeal joint, and Cohen (1948) has summed up the situation by saying, "The skin is dry and shiny, of red or mauve tinge, and the neighbouring veins are distended."

There is therefore a fair amount of collateral evidence that locally increased vascularity, with engorgement of neighbouring veins, is at least a regular concomitant of the suddenly developing swelling. Perhaps what seems to be an almost universal modern tendency to allude to the swelling as "inflammation" is rather to be deplored, since inflammation is a term of rather vague connotation.

There is probably some significance to be attached to the observation so often recorded that, at any rate at some stage in the progress of the attack, the area is not only red and swollen but it is also *dry*. Boerhaave had as an aphorism that "he had never known the gout attack anyone while he continues to perspire" in the feet. Of the sufferer he observes, "His perspiration is checked, especially that of his feet: first the feet become dry, then varicose." The combination of an upset in the circulation of the part with a disturbance in the phenomenon of local sweating is probably of some importance in forming an opinion on the physiological factors involved in the causation of the disease.

Hallmark of the Onset of Gout

When we turn to the subjective aspect of the attack there is no doubt about that: intense pain is the hallmark of the onset of gout. But the subject of pain is a difficult one with which to deal in describing the natural history of any disease, and in gout it is one of more than usual difficulty. An observant physician is able to give a very exact and vividly realistic description of the red and swollen big toe, but when it comes to describing the pain that the patient suffers he is under a severe handicap. In the first place he must needs describe it at second hand: "The patient is the only witness we have, and if he says he has a pain there are no grounds on which we can contradict him" (Sunderland and Kelly, 1949). In the second place, though he may not realize it, he has no adequate language in which to describe it, even should he have ever experienced it in his own person. It is possible that it is these almost insurmountable barriers that prevent some modern clinicians from even attempting to describe the pain attendant on the progress of the disease.

The older generation of physicians sought to give a verbal quality to pain, even if they had to rely on the somewhat crude similes instituted by the patient. We may perhaps disregard their efforts as being, at best, mere word pictures of something not actually realized and so of necessity retailed at second hand and often in the language of persons lacking perhaps both imagination and education. But I think it is beyond doubt that there was a certain flavour about the writings of a former generation of physicians who dealt with the natural history of disease that is lacking in most modern and doubtless more rigorously scientific works. "The patient complains of pain" is a poor colourless substitute for the vividly descriptive phrases that some of the masters of medicine employed.

Classical Descriptions of the Pain

For the moment we will dismiss the handicap that pain must inevitably be described at second hand, for there is a far greater disability imposed by the innate methods of human thinking. I do not wish to labour a point to which I have so often called attention in the past: that man from his first using and fashioning tools and weapons, and on to the phases of constructing simple mechanical devices, to his perfecting a myriad of complicated machines, has always possessed a mechanistic and material imagery and so a mechanistic vocabulary and form of expression. We simply have to face the fact, as all biologists have, that we have no language in which to describe biological or psychic processes. This lack of appropriate language is made evident if we take some of the classical descriptions of the pain felt in an attack of gout. It has been described as resembling the "gnawing of dogs" (Sydenham, Ring, and Garrod), and this seems to be the description most often used.

We all know the story of the medical student who, in the course of an examination, when asked why he had (correctly) diagnosed a swelling as being a varicocele, replied with justifiable confidence: "Because, sir, it feels like worms in a bag." To which the examiner replied with the disconcerting question: "And pray, sir, when last did you feel worms in a bag?" It would be easy to pose the equally embarrassing question to the sufferer from gout: "And pray, sir, when did you last feel dogs gnawing your big toe?" The same criticism applies to the description that the pain resembles a "dislocation of the joint" (Sydenham and Ring), that it is like "tearing the joint asunder" (Garrod), and the many other similes with which the literature abounds. No fault lies with the describer, be he sufferer or physician: the defect is in the fact that, in the absence of a biological vocabulary, recourse must be had to crude physical similes dictated by imagined physical experiences.

Perhaps a nearer approach to truth is the dictum of the Frenchman, quoted by Sir Thomas Watson (1843): "Place your joint in a vice and screw the vice up until you can endure it no longer—that represents rheumatism; then give the instrument another twist and you will obtain a notion of gout." That is certainly the most witty and, in its way, perhaps the most realistic description of gout, but it lacks one thing: it lacks a consideration of the *quality*, as distinct from the *intensity*, of the pain. Gout and rheumatism are as distinct in the quality of the pain that they produce as two pathological conditions can well be. We may well agree with Garrod that "the pain is intense" and with Dyce Duckworth that "the pain increases to positive agony"; but the real secret is revealed in Garrod's further statement that "it is different from other pains." It may be that the first clue to the difference is revealed in the oft-repeated descriptions of gouty patients as being "choleric and irascible" (Ring) or "querulous and irritable" (Cadogan) during the attack: for these expressions indicate in some measure the strangely affective, almost psychical, quality of the pain. As Scudamore (1823) wrote: "The agitation of the mind is much more remarkable [in gout] than in any other inflammatory disease."

A man with sciatica may suffer acute pain, but once he has learned the difficult art of changing position in the manner that produces the minimum of pain he is far on the road to being able to regard the complaint with some equanimity. He dreads the advent of a sneeze; he does not wish to be made to laugh; but beyond that he has no particular anxieties. Sciatica is indubitably a very painful

complaint, but it is one that can be faced with perfect psychic composure.

It is far otherwise with gout: from the first twinge in the big toe a dreadful apprehension overwhelms the patient. He fears lest anyone should come near him and thoughtlessly touch him, or jar him with their footfall, or cause him to move. He dreads the presence of anyone who moves hurriedly, and is terrified lest the fussy person should slip, or drop something, or give him a start of any kind. And in all this it is to be remembered that it is not the actual touch, the jar, or the change of position that causes the pain, it is the apprehension that it might happen. Although there is local pain, enough and to spare, it is the psychic state of dread that characterizes an attack of gout. The initiation of an exacerbation may be caused almost as readily by a mental concept as by physical contact. I do not know the original source of the account of the gouty patient who, having seen a fly upon the ceiling, lay in an agony of anticipation lest it should lose its hold and drop in the neighbourhood of his gouty limb. The picture is doubtless an exaggerated one, and yet it contains a very real element of truth.

Sensation of Heat or Cold

The sensation of local heat that is commonly a pronounced feature of the attack is apparently something altogether apart from the "gnawing" pain. It is not even inevitable that the sensation of heat is present on all occasions, and Sydenham, in describing his own case, said that "the parts feel as if cold water were poured over them." It would seem to be a feature of podagra, as of other conditions that appear to be allied to it, that in the matter of the sensation of heat or cold there are fluctuations during the progress of the attack and differences in individual attacks. Even when the burning sensation is at its height it is obviously a superficial business: it is a cutaneous sensation of heat. The part feels as though the skin were exposed to actual fire and was about to be scorched.

The real and characteristic pain is a deep-seated pain, situated, not in the skin, but deep in the tissues of the affected part. The essential features of this pain are that it is a live, active, and ever-moving pain. As Sydenham said: "Now it is a violent stretching and tearing of the ligaments—now it is a gnawing pain and now a pressure and tightening." One thing only needs to be added to this very realistic description: it is a *radiating* pain. Twitchings, contractions and relaxations, throbbings, pulsations, and flutterings run up and down from the focal point of maximum pain, pulling, tearing, and crushing as they go. Although these feeble descriptive terms are the only ones it is possible to employ, it must be remembered that, though they apparently convey no more than somewhat innocuous connotations, each one expresses a quality that is vividly, even excruciatingly, painful in an attack of gout.

Final Stages

The rest of the story of an attack of periarticular gout is easily told. After a varying interval the pain ceases to be paroxysmal in character and it becomes less in intensity. Some pain may linger for a very long while after the cessation of the acute attack, and after long-continued attacks the normal neutrality of sensation is never restored. Following the cessation of the more acute but non-paroxysmal pain a period of itchiness of the part generally supervenes. This is followed by desquamation of the superficial layers of the skin over the affected area, and it terminates in what can only be described as the "wooden-toe" stage. This final episode consists in the subjective sensation that the part is hardly a living appendage of the body, but is a more or less insensate extension, the presence of which is realized rather by its annoyingly obtrusive character than by its active participation as a functional member. In the case of larger joints, such as the knee, this subjective feeling of artificiality—and so of unreliability—may persist for years after the initial attack.

So much by way of a personal and probably very biased account of the progress of an attack of acute gout. It remains to be seen if the various phases can be fitted together into a composite and more or less reasonable picture of a definite pathological entity.

Support from Authority

Leaving aside, for the moment, the whole question of the part played by some disturbance of the metabolism of uric acid in the causation of gout, it is worth while to inquire what support from acknowledged authority may be found for some of the other conclusions we have arrived at. There is probably no need to quote further evidence in support of the contention that acute gout is essentially a periarticular and not an interarticular disease and is not primarily an arthritis of any kind. That non-specific articular changes often occur in the course of the disease seems to be a well-established thesis in British medical literature: but that any form of arthritis is primarily, or even essentially, a part of the disease is certainly not a proved fact. In my own case the conclusion that "dans l'accès de goutte du gros orteil, la radiographie montre un interligne metatarsophalangien absolument normal" is certainly correct.

That the swelling is due to a change in vascularity and not to an accumulation of exuded humours or peccant matter has been so often asserted that it may be claimed as being the orthodox view. Sydenham (1683), Caverhill (1769), Ring (1811), and Scudamore (1823) have all testified to the periarticular vascular changes, and de Gennes (1935) has summed up the question by saying: "Sur l'accès lui-même, tout est dit, et nous insisterons seulement sur le signe de la distension veineuse que notre maître, le professor Widal, donnait comme le plus fidèle témoin de la goutte." Moreover, two of these observers, Caverhill and Scudamore, formulated theories to account for the curiously localized vascular congestion.

That gout is in essence a disease that affects the nervous system is the whole underlying thesis of Dyce Duckworth's paper published in 1880. In support of this thesis he quotes the previously expressed opinions of Stahl (1737), Laycock (1864), Meldon (1872), Cullen (1872), Liveing (1873), and Paget (1879). He contends "that the diseased conditions which are recognized as of unequivocally gouty nature are *primarily* dependent upon a functional disorder of a definite tract of the nervous system, and that thus gout is a primary neurosis." In his great *Treatise on Gout*, published in 1889, the thesis is modified to the extent that more importance is assigned to the underlying metabolic change; for he sums up by saying: "I venture, then, to suggest that a *secondary* affection of some nerve-centre occurs as a consequence of the altered blood state *ab intra*, and that thus the order and particular process of the gouty attack is evolved." The nerve-centre involved was postulated by him as being connected with the vasomotor nerves.

Scudamore before him had been a strong advocate for the recognition of the nervous influence involved in the phenomena, but in his time the vasomotor nerves had not been recognized as neurological entities. If Duckworth

may be regarded as being among the pioneers in maintaining the importance of recognizing the neurovascular element in gout, Lancereaux (1910) and, later, Finck (1926) should probably be regarded as pioneers in drawing attention to the parts played by the sympathetic nerves and the vagus respectively in the production of the typical vascular disturbances.

Now that we are free to discuss the part played by the vasomotor nerves, we need not be concerned with the older theories of Caverhill or of Scudamore. But the ideas put forward by these writers are not devoid of interest. Caverhill ignored the knowledge of the capillary system that was available to him and postulated "cohesions" occurring in the course of the "cutaneous arteries" of the big toe. He pictured the breaking down of these cohesions and the stretching of the arteries "above the cohering point" preceding the break as being the causes of the paroxysmal pain. Scudamore recognized the part played by capillaries, but he describes "the pulsatory action of the minute arteries, which appears to be in part owing to the distension of the contiguous parts operating as a ligature." Both these accounts, it must be admitted, are graphic and logical explanations of the observed phenomena in the necessary absence of any knowledge of arteriovenous communications under the control of the vasomotor nerves.

Capillary Circulation

Knowledge of the capillary circulation came rather slowly into the sphere of practical medicine and surgery. Knowledge of the arteriovenous channels, "canaux dérivatifs," or Sucquet–Hoyer canals, is a thing only of yesterday as scientific discovery is reckoned. From the time (1628) when Harvey postulated the existence of the ultimate communicating channels connecting the arterial and venous systems that were beyond his ken, until they were actually seen and described by Malpighi, was a period of over thirty years. Following Malpighi's discovery of the capillaries, almost another century was to pass before Stephen Hales recognized that these minute channels were subject to changes in their diameter in varying phases of the circulation. It was not until towards the end of the nineteenth century that Charles Rouget studied minutely the mechanism of spontaneous capillary movements.

Rouget's work was the natural sequel to the independent discoveries of Claude Bernard, Brown-Séquard, and August Waller (1851–3) of the existence of specialized nerves—the vasomotor nerves—controlling the terminal radicles of the circulatory channels. With this discovery the ultimate nerve-controlled ramifications of the circulatory system became of clinical importance and many obscure pathological conditions were recognized as being caused by some upset in this neurovascular mechanism. In 1878 Weir Mitchell described his "rare vasomotor neurosis of the extremities," and since that time the medical literature of vasomotor neuroses has become almost unwieldy in its volume.

Apart from Malpighi's capillaries and the universally recognized minute connexions between the ultimate radicles of the arterial and venous systems, other arteriovenous communications were postulated by many writers early in the nineteenth century. These communications were defined as being more direct and more open than the minute and tortuous channels of the capillaries. They were claimed to be pre-capillary arteriovenous communications. Sucquet, in 1862, seems to have been the first anatomist who claimed to have demonstrated the existence of these "canaux dérivatifs" or pre-capillary arteriovenous communications. He described them as being present in many parts of the body "notamment dans le derme sous-unguéal, la peau des faces plantaire et palmaire et des articulations."

Little attention was paid to Sucquet's work, as it seems to have been rather generally accepted that his observations were erroneous. However, in 1878 Hoyer demonstrated by injection technique that viscous solutions incapable of penetrating the capillary bed could pass direct from artery to vein in those situations in which Sucquet had described his "canaux dérivatifs." It still awaited the work of Masson (1924–37) on the pathology of the so-called glomic tumours before the clinical importance of pre-capillary arteriovenous connexions was widely appreciated. From 1930 R. T. Grant, working in the laboratory of Thomas Lewis, demonstrated, by a very beautiful technique, the physiology of the arteriovenous anastomoses in the ear of the living rabbit. To-day these pre-capillary channels, situated in distal parts, are well recognized as being nervously controlled mechanisms, developed in connexion with regulating the loss or conservation of heat.

Heat-regulating Mechanism

One element of this heat-regulating mechanism is possibly of some importance in the study of gout. The arterial anastomoses around joints are among the commonplaces of topographical anatomy, and in their general disposition they fulfil Hart's law, that "anastomoses are more frequent between arteries in proportion to the remoteness of these vessels from the heart." The anastomoses around the joints become freer and more complex as they are traced distally in the limb, and that in connexion with the big toe is probably the most ample of them all.

Although the periarticular arterial anastomoses are so well known, in consequence of their surgical importance, the corresponding plexuses of periarticular veins receive scant attention in works on descriptive anatomy. Nevertheless, the venous periarticular plexuses of anastomotic vessels are more abundant and more complex than those of the arteries. Moreover, connecting these two complex systems there are the "canaux dérivatifs" described by Sucquet. It is this arrangement that permits each segment of the limb to behave as a self-regulating circulatory unit: arterial blood may be short-circuited into the venous system in the region of every joint by means of the arteriovenous channels.

In very crude fashion the mechanism may be likened to a centrally heated hot-water system, with radiators interposed in the circuit at definite intervals, represented by the joints. The outgoing hot water may pass to the terminal (digital) radiators in full spate and heat may be radiated from the distal points. Or it may be that the terminal radiators are more or less closed down, or that the flow through the more proximal radiators is increased. All these things are made real from homely experiences. The trivial phenomenon of "dead" bloodless fingers slowly recovering and flushing, joint by joint, to the accompaniment of a certain amount of pain as the blood supply returns is familiar. Perhaps a more homely manifestation of this same regulating mechanism is within the experience of everyone, for it is probably a universal grievance that long after the body has got comfortably warm in bed the feet remain persistently cold.

A far graver and more impressive expression of the same physiological phenomenon is the centripetal advance, joint by joint, of the progress of dry gangrene in certain cases. Anyone who has witnessed such a march of dry gangrene in the lower limb of a human subject following ligation of the main artery, or has seen similar conditions in lower animals, must realize that at the periarticular anastomoses

and short-circuits of arterial and venous blood a definite and sharp-cut boundary-line exists between the circulation of one segment of the limb and that lying immediately proximal to it.

In a perfectly normal human foot in fresh condition and on which no hardening or preservative reagents had been used, injection was made into the popliteal artery. The injected material was a viscous basis composed of gelatin, and in the mass was incorporated a particulate colouring matter (vermilion). Within a few seconds of the arterial injection being started the big toe flushed pink, and during the next few seconds the superficial veins became engorged and the injection mass returned from the veins at the cut surface of the amputated part. The resulting specimen is shown in Fig. 3, and it demonstrates dramatically the freedom of the passage of even a viscous injection mass (carrying suspended solid particles) from the arteries to the veins in the region of the big toe.

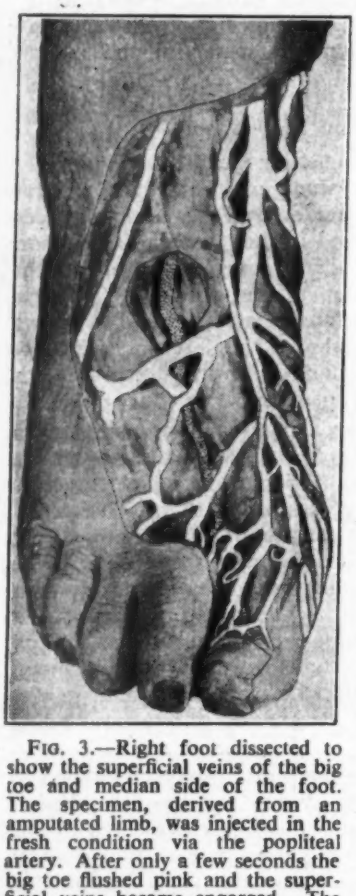

FIG. 3.—Right foot dissected to show the superficial veins of the big toe and median side of the foot. The specimen, derived from an amputated limb, was injected in the fresh condition via the popliteal artery. After only a few seconds the big toe flushed pink and the superficial veins became engorged. The only arterial channels shown in the dissection are part of the dorsalis pedis and the first dorsal metatarsal artery running to the big toe. The arteries are stippled.

It is a simple (perhaps too simple) matter to picture that some vasomotor upset of this very obvious anatomical vascular arrangement normal to the big toe is responsible for the symptoms of podagra, and that the similar conditions prevailing in the periarticular anastomoses of other joints account for the less frequent manifestations of so-called "articular" gout. If this be so, certain other possibilities are opened up. Perhaps periarticular, or classical, gout might find its rightful place among such an assemblage of vasomotor syndromes as Weir Mitchell's erythromelalgia (erythermalgia), causalgia, Raynaud's phenomenon, acrocyanosis, pernio, and the many other conditions in which autonomic nerve involvements in the limbs are the outstanding manifestation. There is certainly an almost wearying likeness in the descriptions given by patients, and even by clinicians, of the subjective and objective symptoms of these several pathological conditions. "The pain is throbbing, aching, and burning. . . . Usually it comes in the ball of the foot or of the great toe," which is in a state of "mottled redness as if the smaller vessels were always over-distended." The "pain felt, he said, as though barbed wire were being pulled through the foot. . . . Seeing a man on crutches trip over, or seeing two motor-cars nearly collide . . . brought on exacerbations.

The first of these descriptions is taken from Weir Mitchell's account of a case of erythromelalgia, the second from Sunderland's paper on causalgia, and both seem to evince a family likeness to the classical descriptions of an attack of gout.

Two Questions

It would indeed seem that Duckworth's contention that classical gout is a form of neurosis affecting the "nerve-centre" of the vasomotor nerves might have a very considerable element of truth in it. If this indeed be so, two questions remain for answer: "What causes the disorder of the vasomotor nerves?" and, "What, if any, is the action of colchicum on the neurovascular mechanism?" The answers to these questions lie naturally without my province; but certain statements in the literature are not without interest.

Duckworth claimed that the "affection of some nerve-centre occurs as a consequence of the altered blood state *ab intra*." By that he presumably meant that the disordered metabolism of uric acid was the cause of the nerve involvement. H. Frédéricq (1923) has stated very definitely that purines act upon the sympathetic system. Finck (1926) strongly supports him and quotes other authorities, such as Palay, who have investigated the effect of purines on the sympathetic nerves. There, unfortunately, the question must be left by an anatomist who has trespassed outside his legitimate field.

As for the action of colchicum, I believe that even an anatomist may have a word to say from the practical experience of having taken large quantities of it. From Garrod (1859) to Cushing (1947) there is complete agreement that colchicum has no action whatever on uric acid metabolism: but with the passage of time there is a curious change of opinion concerning its action on vasomotor nerves. Duckworth sums up the action of colchicum by saying: "The peculiar benefit derived from this drug is not secured in any other form of inflammation, and this is plainly specific. Its cherished action is doubtless exerted upon the vasomotor nerves." He quotes Meldon and others, who, having tried the effect of the drug upon themselves during health, experienced "a general glow at the surface of the body, diaphoresis, throbbing of the blood vessels and palpitation." Bence-Jones in 1867, stated that colchicum "acts on the nerves of the capillaries, stopping the inflammation of gout." Continental physicians held exactly the same views; but apparently no recent authority on pharmacology credits this action to colchicine. It seems possible that there may be some difference in action between the old preparations made from the corm or the seeds of *Colchicum autumnale* (of which some mercifully persist on the market as "patent medicines") and the pure colchicine available during the last twenty years or so.

If it should ultimately prove to be the case that classical or regular gout is in fact a syndrome connected with some disturbance of the vasomotor nerves controlling the periarticular plexuses, a further interest attaches to its sex incidence. That it is almost exclusively associated with masculinity is an observation dating from the time of Hippocrates. So much importance has been attached to this fact that at one time castration was recommended for its cure. On the other hand, Raynaud's phenomenon, which in so many of its features is the antithesis of classical gout, is (apart from industrial vibratory trauma) essentially a syndrome manifested in females. The study of the inheritance of these two conditions obviously opens up an interesting field to the geneticist; and some scattered facts are already available to form the basis of such a study.

Conclusion

I have tried hard to keep the vow with which I prefaced my remarks, and I have been careful to trespass as little

as possible outside the sphere of my own experience. As a consequence I am well aware that I have not discussed the general problem of gout. I know well that the condition that I have been discussing under the name of regular or classical gout is not the only disease passing under that name. I have not spoken of arthritic gout, or tophi, or chalkstones, or deposits of urate of soda. The clinical entity that is distinguished by these characteristics seems to be a lineal descendant of the rheumatic gout of the early nineteenth century that was so graphically portrayed by Adams.

The relationship of the arthritic condition to the spasmodic vasomotor disturbances manifested in classical or periarticular gout can be determined only by the clinician. Since all scientific nomenclature depends upon the question of priority, it seems clear that the vasomotor syndrome may rightly claim the name of gout; and some other name should be given to the gross destructive arthritic condition—unless it can be clearly shown that this is the definite sequela of a previous periarticular vasomotor disturbance.

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The Queen's Institute of District Nursing has issued a pamphlet entitled *Houses for District Nurses*, describing the type of house and district room recommended by the institute as most suitable for district nurses and midwives. It has been prepared in response to inquiries from a number of medical officers of health and nursing associations. The plan and appearance of the house are depicted, accompanied by a short description. Copies may be obtained for 1s., post free, from the institute at 57, Lower Belgrave Street, London, S.W.1.

SIGNIFICANCE OF HAEMOGLOBIN LEVEL IN THE PUERPERIUM

ITS RELATION TO BIRTH WEIGHT AND PRODUCTION OF BREAST MILK

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There have been innumerable studies of haemoglobin in pregnancy, and some have been continued into the puerperium. Very few, however, have included an attempt to assess quantitatively the value of high or low levels, and the advantages of a high level have usually been assumed without extensive proof. One such attempt was made by Reid and Mackintosh (1937a) in their analysis of data obtained in Stockport. They placed about 1,000 women in two groups according to their economic circumstances, and found that when a low level of haemoglobin during pregnancy was associated with poverty it apparently led to an increase in the neonatal and infantile death rates and in the amount of illness in the first year; the period of breast-feeding was also likely to be curtailed. A low level of haemoglobin did not seem to bring similar disadvantages to the higher-income group.

The present work consists of an analysis of the results of about 1,500 haemoglobin estimations made on women who were delivered in the Landesfrauenklinik der Rhein-Provinz, Wuppertal, between June and December, 1946. The estimations were carried out on the seventh day after delivery, and always by one or other of two hospital laboratory assistants. The Sahli method was used, and the colour was assessed in an optical haemoglobinometer; 100% on the scale of this instrument was said to represent 14 g. of haemoglobin. The nutritional status of the women is discussed in full elsewhere (Dean, 1950), and in the same place it is reported that in the years when the food supply was reduced—which included 1946—the children were smaller at birth than children born in times of plenty, and mothers had much more difficulty in establishing lactation. There was no rise in the incidence of any of the diseases of pregnancy or the puerperium, and the children appeared to be normal in every way except size. The mothers were not given iron during pregnancy. Their diets contained large quantities of vegetables and therefore adequate amounts of vitamin C. There were no signs of vitamin deficiencies and none of the women had oedema of nutritional origin.

Unfortunately, estimations of haemoglobin had not previously been done as a routine at the Landesfrauenklinik, and it was therefore impossible to make comparisons with the pre-war years. Cases of multiple pregnancy, caesarean section, and intercurrent illness have not been included in the analyses; cases of post-partum haemorrhage have been considered separately in the sections dealing with lactation but have otherwise been omitted. It was the custom to weigh, and not merely to estimate, the blood loss at delivery.

Results

General.—The distribution of women with high and low levels of haemoglobin is shown in Table I. If the women who lost 500 g. of blood or more at delivery are omitted, more than 20% of the total number were in the range of haemoglobin up to 77% and less than 80% were in the higher haemoglobin range.

TABLE I.—*Distribution of Women with Different Levels of Haemoglobin on the Seventh Day After Delivery*

	Haemoglobin Per Cent							Total
	Up to 50	51–59	60–68	69–77	78–86	87–95	96 and Over	
No. of women, including those who had a post-partum haemorrhage* ..	7	36	131	252	477	480	121	1,504
Percentage of total	0·05	2·4	8·6	16·7	31·7	31·9	8·7	100
No. of women, excluding those who had a post-partum haemorrhage ..	3	17	76	209	440	464	118	1,327
Percentage of total	0·02	1·3	5·7	15·8	33·3	35·0	8·9	100

* Defined as loss of 500 g. of blood or more.

Relation of Haemoglobin to Yield of Milk on the Fifth Day.—In Table II the mean percentage of haemoglobin of women who provided different amounts of breast milk on the fifth day is given. The general ability to lactate was poor, less than half of the women being able to provide more than 250 g. The milk yield was not related to the mean percentage of haemoglobin, the small differences in the means not being statistically significant. The separation of the women into those who had haemorrhage and those who had none emphasizes the fact that lactation was no worse at the 68–74% level of haemoglobin than at the 82–85% level. These results are perhaps surprising, but they are in accordance with the findings of Reid and Mackintosh (1937b).

TABLE II.—*Relation of Milk Yield on the Fifth Day to the Mother's Haemoglobin on the Seventh Day and the Effect of Post-partum Haemorrhage**

Yield of Milk on 5th Day (g.)	Women Who Had No P.-p.h.				Women Who Had P.-p.h.			
	No. in Each Group	Percentage of Total	Mean Hb %	S.D.	No. in Each Group	Percentage of Total	Mean Hb %	S.D.
Up to 100 ..	77	5·4	82·8	10·9	10	0·7	71·8	8·3
110–150 ..	122	8·6	84·4	9·3	23	1·6	73·8	14·7
160–200 ..	234	16·5	83·8	9·0	36	2·5	68·3	10·4
210–250 ..	257	18·1	84·4	9·5	38	2·7	72·7	10·8
260–300 ..	245	17·2	84·4	9·4	22	1·6	73·6	11·2
310–350 ..	142	10·0	84·2	7·4	20	1·4	71·2	11·5
360 and over	167	11·7	84·8	9·0	28	2·0	70·0	12·5
Total ..	1,244	87·5			177	12·5		

* Defined as loss of 500 g. or more.

Effect of Large Post-partum Haemorrhage.—Forty five women lost at delivery amounts of blood said to exceed 1,000 g. The effects of these losses are shown in Table III, in which primiparous and multiparous women, and those with male and female children, have been dealt with separately. For each set, controls have been provided by taking all the available records of women who gave birth in the clinic in the same period to children of the same birth weights as the averages but who had no haemorrhage during delivery. It is clear that although the haemorrhages probably caused a reduction of about 20% haemoglobin, they did not affect the lactation on the fifth to eighth days. König (1939) also found that haemorrhages of between 500 and 1,000 g. seemed to have little effect on early lactation.

Relation Between Birth Weight of the Child and the Mother's Haemoglobin on the Seventh Day.—The evidence of Table IV suggests that there was no correlation between the size of the child and the haemoglobin level of the mother. The lowest haemoglobin concentration—81.1%—was that of the mothers of the smallest children, but the difference between this and any of the other mean concentrations is not statistically significant. Reid and Mackintosh (1937a) were also unable to find any relation between the level of the mother's haemoglobin and the size of her child. If deliveries with post-partum haemorrhage exceeding 500 g. had been included in Table IV the average haemoglobin levels for the heavier weight groups, but not for the lighter groups, would have been considerably lower. In accordance with usual clinical experience most of the haemorrhages occurred where the children were above the average in weight.

TABLE IV.—*Relation Between the Birth Weight of the Child and the Mother's Haemoglobin on the Seventh Day*

Birth Weight of Child (g.)	No. in Each Group	Percentage of Total	Mean Hb %	S.D.
Up to 2,000	35	2·6	81·1	10·4
2,001–2,400	29	2·2	83·5	11·8
2,401–2,800	140	10·5	84·8	9·1
2,801–3,200	410	30·9	84·0	9·5
3,201–3,600	429	32·3	84·4	9·2
3,601–4,000	209	15·8	83·4	9·6
4,001 and over ..	75	5·7	82·8	9·5
Total	1,327	100·0		

Cases of post-partum haemorrhage of 500 g. or more have not been included.

Relation Between the Parity of the Mother, the Sex of the Child, and the Mother's Haemoglobin.—Mothers who had already had a pregnancy and those who gave birth to female children had slightly higher haemoglobin levels on the seventh day than the primiparae and mothers of male children (Table V). The differences in the means are small

TABLE V.—*Relation of the Level of the Mother's Haemoglobin on the Seventh Day to Her Parity and the Sex of Her Child*

	Mothers of First Children	Mothers of Later Children	Mothers of Boys	Mothers of Girls
No. in group	666	661	686	641
Average birth weight of children (g.) ..	3,130	3,295	3,260	3,150
Mean Hb % on 7th day	83·3	84·5	83·4	84·5
S.D.	9·1	9·3	9·4	9·3

Women who had a post-partum haemorrhage of 500 g. or over have not been included.

TABLE III.—*Effect on Lactation of Post-partum Haemorrhage Exceeding 1,000 g.*

Parity of Mother	With or Without Haemorrhage	No. in Group	Child		Mother							
			Sex	Average Birth Weight (g.)	Average Age (Years)	Average Hb %	Milk Yield (g.)				Standard Deviation	
							5th Day	6th Day	7th Day	8th Day	Hb	5th Day Milk
Primiparous ..	With	10	M	3,400	27	64	270	325	330	350	9·7	88·7
	Without	21	M	3,400	27	82	250	300	325	340	8·0	71·1
Multiparous ..	With	12	M	3,500	31	66	235	260	290	310	7·7	88·9
	Without	16	M	3,500	31	88	240	285	310	325	7·3	99·4
Primiparous ..	With	9	F	3,050	30	68	250	270	290	360	5·8	102·0
	Without	12	F	3,050	25	87	217	242	281	290	8·6	57·6
Multiparous ..	With	14	F	3,300	33	65	295	300	355	385	6·6	55·0
	Without	13	F	3,300	29	92	252	294	303	315	4·8	92·6

and possibly not of biological importance, but are nevertheless significant according to statistical definition (being more than twice the standard error of the differences). Roscoe and Donaldson (1946) and Scott and Govan (1949) could detect no difference due to parity, but they were reporting comparatively small series of results of estimations done at various stages of pregnancy.

Relation Between the Age of the Mother and Her Haemoglobin Level.—This relation was examined because it has been suggested that age might be a factor in determining the onset of anaemia in pregnancy (Roscoe and Donaldson, 1946). It will be seen from Table VI that the mean haemoglobin on the seventh day was not related to the mother's age in the present series. There was a similar lack of correlation in the data of Reid and Mackintosh (1937b) and Scott and Govan (1949).

TABLE VI.—*Relation Between Age of Mother and Haemoglobin Level in Her Blood on the Seventh Day After Delivery*

	Age in Years			
	Up to 23	24–29	30–35	36 and Over
Total number of mothers in each group	295	479	327	226
Mean Hb %	83·9	84·0	84·8	83·1
S.D.	9·8	9·3	9·1	10·1

Women who had a post-partum haemorrhage of 500 g. or more have not been included.

Discussion

The significance of the haemoglobin level in pregnancy and the puerperium is complicated by the changes that occur in the dilution of the blood. The hydraemia was noticed in the data of Denis (1830) by Nasse (1836) and was investigated by Andral and Gavarret. The work of the last-named was reported by Cazeaux (1845) in the second edition of his *Traité de l'Art des Accouchements,* and in the third edition (1850) there are summaries of several studies which together establish not merely the hydraemia but the fall in total solids, in protein, and in total blood iron. Willcocks (1881) noticed that the changes in the blood became more pronounced as pregnancy advanced, but the significance of this observation, which has since been repeatedly confirmed, appears still to escape general recognition ; papers are still being written in which haemoglobin levels in pregnancy are reported without proper regard to the time in the pregnancy when the samples were taken.

McLennan and Thouin (1948) critically reviewed the previous work on the measurement of the hydraemia and decided that it was inconclusive. They reported that at term the plasma volume, estimated directly by a dye method and expressed in relation to body weight, was about 40% higher than in the non-pregnant woman, the total blood volume about 32% higher, and the red cell volume 20% higher ; and they thought that a week after delivery the blood volume was probably back to the non-pregnant level. They said, however, " It appears unlikely that anything yet published, including this report, has given a thoroughly reliable picture of what happens to the blood volume in pregnancy."

Mull and Bill (1941, 1945), who estimated the concentration of various elements of the blood, described a process of haemodilution that began soon after conception and continued until about the 35th week. They found that from then until the birth of the child the concentration of the plasma rose and that the rise went on into the puerperium. Their findings confirm the changes in the mother's weight described by Stander and Pastore (1940). The weight increases, of which the greater part can probably be accounted for by hydraemia, continue until about the 35th week. From then until a week before delivery there is a further rise, but this must be largely due to the growth of the foetus, which is very rapid at this time (Hamilton, Boyd, and Mossman, 1945). During the week before the birth the weight of the mother falls, and this fall apparently represents a large reduction in her water load, since it occurs despite the increase in weight of the foetus.

The haemoglobin level on the seventh day after delivery is probably closely related to the antecedent blood levels, but is the resultant of a number of remarkable changes. Some of these are represented graphically in the Chart, from which it seems that on the seventh day the blood concentration is probably about the same as it was at the beginning of pregnancy, although the mother's weight is still about 10% higher than it was then. The Chart also shows that the diagnosis of anaemia in pregnancy should never be made without consideration of the expected degree of hydraemia : this may easily be responsible for the apparent loss of well over 1 g. of haemoglobin per 100 ml. (Widdowson, 1939 ; Committee on Haemoglobin Surveys, 1945 ; Roscoe and Donaldson, 1946). The appreciation of this fact, and the present demonstration that within very wide limits the haemoglobin level in the blood of the mother is unlikely to affect the birth weight of the child or the ability to lactate, might perhaps lead to some restraint on the present-day tendency to give iron lavishly to the pregnant woman. The results, so far as raising the level of haemoglobin is concerned, are in practice often disappointing, and little is really known about the benefits of the therapy except possibly in cases of extreme anaemia. If the work on pigs done by Venn, McCance, and Widdowson (1947) is applicable to man, it is unlikely that the administration of iron to the mother will make any difference to the store of iron in her baby at birth.

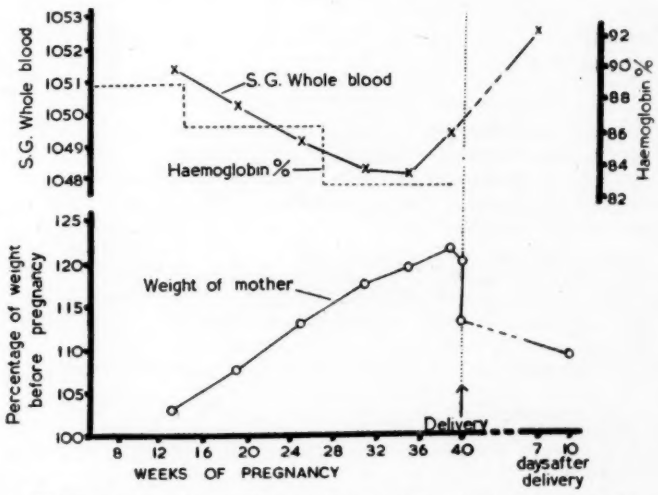

Chart showing the specific gravity of whole blood (Mull and Bill, 1945), the haemoglobin percentage (Haldane—Committee on Haemoglobin Surveys, 1945), and the weight changes (Stander and Pastore, 1940) during pregnancy.

It seems also that even fairly large post-partum haemorrhage has no effect on early lactation and probably, because the fifth-day yield is such a good indication of future success (Robinson, 1947), none on later lactation. The blood lost is diluted blood ; Willcocks (1881) spoke of " the enormous loss of blood from which some women suffer during parturition with comparative impunity," and pointed out that dilution would offer an explanation of the impunity.

We have not done much more than establish his hypothesis a little more firmly.

The explanation of the higher level of haemoglobin found in multiparae may be that although primiparae and multiparae undergo similar weight changes in pregnancy (Stander and Pastore 1940) the primiparae possibly tend to return more slowly towards their original weight; their blood may therefore be a little more dilute on the seventh day. No satisfactory reason can be offered to account for the difference in the mother's haemoglobin according to the sex of the child. It is not related to the different weights of male and female children at birth, because the weight seems to be without influence on the mother's haemoglobin. It may possibly bear some relation to different amounts of circulating sexual hormones which have some effect on water balance, but this is conjecture which cannot at the moment be supported.

Summary

The results of the analyses of about 1,500 haemoglobin estimations made on women seven days after delivery in Wuppertal, Germany, in 1946, indicated that there was no correlation between the haemoglobin level and the yield of breast milk on the fifth day after delivery; that post-partum haemorrhage did not affect the yield of milk on the fifth day; that the haemoglobin level bore no relation to the size of the child at birth; that multiparous mothers and those with female children had a slightly higher haemoglobin level than primiparous mothers and those with male children; and that the haemoglobin level did not vary with the age of the mother.

My warmest thanks are due to Professor Anselmino, Director of the Landesfrauenklinik, for the co-operation which enabled me to carry out this investigation. The collection of the data was largely the work of one of his assistants, Dr. R. A. von Finck.

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The Report for 1948 of the Pest Infestation Research Board of the Department of Scientific and Industrial Research has recently been published (H.M.S.O., 1949, 1s.). It describes work on grain storage and on some major insect and mite pests, particularly the flour mite. There is an account of the work done on insecticides, with special reference to the transfer of D.D.T. from impregnated sacking to foodstuffs. Insecticidal smokes have been studied, and further work has been done on the control of blowflies in slaughter-houses and on the use of methyl bromide in silo bins. Some preliminary investigations have been undertaken on apparatus and methods which will be employed for isotopic tracer studies.

RESEARCH INTO THE ORIGINS OF DELINQUENT BEHAVIOUR*

BY

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Research into the origins of juvenile delinquency, urgent and necessary though it is for its own sake and to the problems to which this paper is directed, is but one of several approaches to the scientific study of co-operation and strife amongst mankind. It may well be that a full understanding of the forces at work in our chosen field will illumine problems of even greater social gravity. However this may be, there can be no doubt that the present prevalence of juvenile delinquency and our impotence to prevent it demand much greater understanding of the forces responsible for it. The purpose of this paper is to explore some of the principles which must be borne in mind in framing research into its origins and to give especial attention to those factors which lie within the child's family life and personality.

Basic Assumptions

Much research into delinquency in the past has been vitiated by three false assumptions: (1) that delinquency is something clear-cut and distinct from other forms of behaviour; (2) that there is no necessity for qualitative distinctions, and that all cases can be dealt with as though they were essentially similar; and (3) that the problem can be studied by concentrating on cases brought into court.

These three assumptions have much in common, and to some extent support each other. As Lewin (1931) has shown, they are characteristic of the pre-scientific phase in the study of a subject and are reminiscent of the days when physicists thought in terms of heat and cold instead of degrees of heat, of lightness and heaviness instead of in terms of mass and gravity.

Perhaps the point can best be illustrated by an analogy from the field of nutrition research. If the same assumptions were to be made, we should believe: (1) that malnutrition was something essentially different and could be clearly separated from good nutrition; (2) that all cases of malnutrition are essentially similar and that there is no need to distinguish one kind from another; and (3) that the problem can be studied satisfactorily by concentrating on cases brought into hospital.

All three are clearly absurd. If the assumptions which actually underlie nutrition research are adopted in the study of delinquency we should accept the following:

1. That the extent to which any of us conform with the social and criminal codes is a matter of degree. There are no blacks or whites, only shades of grey.

2. That those who violate social and criminal codes do so for very many reasons, and that before studying our cases we must do our best to group them into different sorts. Such a sorting must not be done in terms of the individual's offences. The relevant criteria for sorting are twofold: (*a*) those relating to the individual's personal make-up and his capacity for making relationships to his fellow-men, and (*b*) those relating to the social structure of the community and its capacity for making relationships with him.

3. That we must not confine our attention to those brought before the courts, but must investigate all cases of uncooperative and antisocial behaviour wherever found.

*Based on a paper read to a conference on the Scientific Study of Juvenile Delinquency at the Royal Institution on October 1, 1949.

Before proceeding further it is necessary to comment on the planning of research. Research is of two kinds, the unplanned and the planned. Though many of the most fruitful hypotheses are born to individuals working without a set plan, hypotheses can rarely be tested for validity in such conditions. The purpose of planned research is to put promising hypotheses to the test, and, since these are not lacking in our chosen field, research proposals which do not clearly state the hypotheses to be tested cannot be taken seriously, for projects so based are likely to be stillborn.

Note on Social Factors

It will be realized that these assumptions carry with them far-reaching implications. In the first place, if we deny the scientific validity of "being charged in court" as a criterion of delinquent behaviour and extend our inquiry to "all cases of uncooperative and antisocial behaviour wherever found," we not only extend our sphere of inquiry but may call in question the behaviour of citizens who, because they obey the written law, have hitherto been regarded as "good." For instance, with juveniles we have to consider whether the behaviour of parents and teachers who use severe disciplinary measures is to be regarded as co-operative or antisocial. Such inquiries may not be to society's liking, but concentration on the so-called criminal to the exclusion of the people with whom he lives is to study only half the problem. It takes two to make a delinquent as it does to make a quarrel.

Studies of the attitudes of dominant social groups and individuals towards the less successful members of the community, or of the effects of different social structures on delinquency rates, have been conspicuous by their absence. Yet both common experience and exact social research suggest that inquiries of this kind are essential. For instance, it is well known in the armed Services that crime rates are a function of the unit's morale, and that this morale in turn is in no small measure a function of the personalities and attitudes of the officers, in particular the commanding officer. To understand crime in a unit it is clearly necessary to investigate the morale of the unit and the personality of its officers as well as the personalities and attitudes of offenders.

The importance of such studies has been emphasized by the classical experiments of Lippitt and White (1947) on the effects of the "climate" of social groups. These demonstrated the way in which the behaviour of 11-year-old boys varied according to the attitudes of their group leaders. The same boys who under a "democratic" leader had been hard working and co-operative became quarrelsome and bullying, either towards other groups or towards each other, when their leader treated them in a dictatorial way.

Enough has been said to show not only that social research into delinquency is a large and complicated field of its own but that it is in serious need of reformulation to take account of factors hitherto neglected.

The Problem of "Susceptibility"

In discussing the importance of influences such as bad companionship, cinemas, pin-table saloons, lack of playing-fields, and so on, Reckless (1943) has emphasized the fact that, despite exposure to these allegedly bad influences, the great majority of children remain well behaved. Why is it that some succumb? "At several critical points in the study of situational factors the question persistently arises as to the differential responses of persons. What is the difference between those who are and those who are not affected? What sorts of individuals are they who succumb and what sorts are they who remain immune?"

Carr-Saunders *et al.* (1942) pose the same problem, using the term "susceptibility" as an equivalent for Reckless's "differential response." "We shall not begin to know what to do with regard to susceptible types," they write, "until progress has been made in this [psychological] field, and unless we have well-founded lines of action in regard to susceptibility we cannot make any attempt to cope with one half of the problem." That it is in truth one half of the problem is indicated by the little-known fact that, of the men and women whom we commit to prison, one-half have been there before and one-quarter are going for at least their sixth time (Report of the Departmental Committee on Persistent Offenders, 1932). Even at the age of 16, one-third of those charged in the courts have been charged before (Carr-Saunders *et al.*, 1942), whilst the Borstal failure rate is about 40% (*Hansard*, February 24, 1947). Thus one-half of the problem is that of a "susceptible-stage" army. Whether or not an individual enlists in this army is clearly a psychological problem—Why is it that some individuals grow up to be more susceptible to antisocial influences than do others?

On the basis of a large amount of clinical work certain working hypotheses regarding "susceptibility" have been framed and underlie most child-guidance work in Britain to-day. It is these hypotheses which should be subjected to rigorous testing. If they prove true, an answer will have been found to one of our central problems, and once they are accepted as a basis for action far-reaching changes in our preventive and penal machinery will be called for. If on the contrary they prove false, much mistakenly directed effort will be saved and time and energy put to better use.

Hypotheses Regarding "Susceptibility"

Research in child development on both sides of the Atlantic suggests that the extent to which an individual makes trusting, affectionate, and co-operative relations with others and the extent to which he develops an effective conscience depend to a high degree on the relationship which he had with his parents, especially his mother, in his early years. This is a many-sided relationship, comprising within it nourishment and care, training and discipline, and companionship in the joys and sorrows of life. Though the shape it takes depends on many external factors—poverty or wealth, housing, health, employment, war, number of children—every study has shown that such factors are subsidiary to the all-pervasive influence of the personalities of the parents. The relationships within a home cannot be understood unless we concern ourselves with such seeming imponderables as love and hate, jealousy, nagging, fairness and favouritism, generosity and charity, loyalty, dishonesty, and self-sacrifice. It is these "springs of action of the human heart" which go to make or mar the family and to shape the characters of the children growing within it.

That the problem of the origin of delinquency is bound up with human feeling and family atmosphere has been emphasized in many of the studies of the past 25 years. In his estimate of the significance of environmental conditions Burt (1925) enumerates defective discipline, vicious homes, and defective family relations as of first importance, and conditions outside the home and poverty as of less importance. Aichorn (1935), describing 12 exceptionally aggressive and delinquent boys, writes: "In every case there was a severe conflict between the two parents

or with the child, so that the child was found to take sides with either the father or the mother or against both. All these children had been brought up without affection and had suffered unreasonable severity and brutality." Healy and Bronner (1936) conclude : "No one finding was so discriminatory between the pairs [of delinquent and non-delinquent siblings] as the fact that at least 91% of the delinquents gave clear evidence of being or having been extremely disturbed because of emotion-provoking relationships with others, mainly with others in the family. We could detect the presence of similar inner stresses in not more than 13% of the controls, and even then to a much lesser degree."

Bowlby (1940, 1944), in comparing a group of 44 juvenile thieves with a control group of other maladjusted children, demonstfated both clinically and statistically that prolonged mother–child separation in the first five years of life bears a specific aetiological relationship to persistent stealing. This finding has been confirmed by Bender (1947), who bases her conclusions on some hundreds of cases of psychopathic behaviour disorders in children. "This group of children presents a clinical picture which forms a syndrome in that the causative factors in the early life of the individual are known, the developmental course may be anticipated, the behaviour pattern is typical and closely resembles the classical description of the so-called constitutional psychopathic personality. . . . The cause of the condition is emotional deprivation in the infantile period due to lack or a serious break in the parent–child relationship. . . . The defect is in the ability to form relationships, to identify themselves with others."

Finally, Stott (1950), who, under the auspices of the Carnegie United Kingdom Trust, has recently been studying a consecutive group of approved-school boys aged from 15 to 18, concludes : "It was discovered in every case that the boy had been conditioned to an anxiety over his parents or to an enhanced emotional dependence upon them by one or more influences operating during his earlier childhood," and instances as being especially prevalent : illnesses in early life, more or less prolonged early separation from the mother, and the irritable character of the mother herself. That Stott, who admits he began his work critical of such conclusions, should have found such factors in every case of a random group of approved-school boys is highly significant. His report, which gives detailed evidence about all his cases, will be published shortly and is likely to become a standard work.

Two Common Factors

Indeed, the evidence that the quality of the young child's human experiences within the family affect his later social behaviour is now too strong to be ignored and demands systematic study. In undertaking this, it is useful to consider as a basic function of the human being his capacity to make affectionate and lasting friendships with others. This function, like vision or digestion, can either develop favourably or be subject to varying types and degrees of disorder. Taking it as our focus, we can proceed to the systematic study of the effects upon its development of different experiences in early childhood. Work to date suggests that, within the main body of hypotheses regarding the child's social development, two factors can be picked out which are especially common in the history of persistent delinquents and differentiate them from children suffering from other forms of maladjustment. These are :

1. Prolonged separation of the child from his mother, or established foster-mother, during his first five years. Such separations are of not less than six months, during which the child is with complete strangers—for example, in hospital, an institution, with unknown relatives, etc. Changes from one mother-figure to another during these years may have the same result. The evidence presented independently by Bowlby (1944) and Bender (1947) supports this hypothesis, and suggests that this one factor alone accounts for a high proportion, perhaps half, of the most intractable cases.

2. A child's being more or less unwanted by parents who are themselves unstable and unhappy people and whose attitudes towards him are, on balance, hostile, critical, and punishing. This hypothesis arises particularly from the work of Healy and Bronner (1936) and of Stott (1950), though it emerges from several other studies—for instance, those of Aichorn (1935) and Bowlby (1944). These unfavourable parental attitudes themselves need explanation. The evidence available shows that they spring from the parents' own unhappy childhood, which has made them prone to a resentful and hostile attitude towards people. Though they recall their unhappy relations with their parents, they are often quite unconscious of the way it is affecting their treatment of their own children. The abuse and criticism so often meted out to these parents is unprofitable and frequently worsens their attitude. Instead they need skilled and understanding help.

Not only is it believed that these two factors would be found in highly significant degree amongst the "susceptible" but on the basis of clinical experience it can be predicted that the children who have suffered these two sorts of experience will as a rule be readily distinguishable from each other, both in their general social relations and in their delinquencies.

It must of course be emphasized that these two classes of delinquent, each with its own aetiology, are not to be thought of as exhausting all types of "susceptible" personality. Apart from mixtures between the two, other classes almost certainly exist, including those of hysterical or depressive make-up whose condition follows a traumatic bereavement in childhood. The two classes described, however, are believed together to account for a majority of the more intractable cases, including the "constitutional psychopaths" and the "moral defectives."

Hypotheses regarding the effects of particular sorts of family experiences on social development can be stated with much greater precision and in far greater detail. It is to be noted that they in no way contradict theories postulating the influence of hereditary factors, except in so far as these are held in the extreme form that hereditary factors alone account for all differences in human behaviour. All that this body of hypotheses claims is that the child's early social environment is relevant in a highly significant degree to an understanding of later social behaviour. Most of those subscribing to this view believe that in the final analysis hereditary factors will also be shown to play a part.

Considerations Regarding Research Procedure

It would be inappropriate in a paper of this kind to attempt the description of particular projects for child-study. There are very many ways in which these hypotheses could be put to the test ; our difficulties arise when we come to select out of the many just the one or two which, with the minimum of effort, will give the maximum of relevant information. The planning of the right project is itself a technical and time-consuming task requiring both theoretical exploration and reconnaissance in the field.

There are certain considerations, however, which apply to any project in this subject, research in which is far from easy. If we are to make a realistic assessment of the child's early social experiences we must take account of very many significant factors and go into them in great

detail. Blanket generalizations regarding "family atmosphere" or "broken homes" are useless. Nothing but a thorough and detailed analysis of the young child's "social diet" is adequate in studies of the origin of co-operative and antisocial attitudes. There are, however, very great difficulties in obtaining reliable data on these factors. Some of these difficulties are inherent in any research which seeks to study the connexions between events occurring many years distant in time from one another. The day will no doubt come when we have reliable and agreed tests for assessing a person's "susceptibility" without resort to the cumbersome and crude method of watching his behaviour over many years of social living—tests which will perform the same function in our field as the Mantoux and the Schick perform in the field of infectious illness.

Meanwhile, if we are to validate our hypotheses, we have no option but to accept as criteria for "susceptibility" the individual's social behaviour and to seek to establish a connexion between that and events which have occurred many years earlier in his life. To achieve this there are two possible procedures—either to follow a group of infants over ten or fifteen years, obtaining first-hand information about their experiences as we go, or else to strive to unearth the past histories of children or adolescents. The first, the current method, has the serious disadvantage that the research has to be carried on over ten or fifteen years before any results are available. The second, the retrospective method, is dependent for half its data being furnished many years later by untrained and by no means disinterested observers—the children's parents.

In the long run the current method has many advantages, and several projects of this kind are being undertaken in the United States. The retrospective method, however, can still be of great value provided it is used with more understanding and insight than has often been the case in the past. For obvious reasons the longer the interval the less reliable the data and the more time required to unearth it. Experience suggests that reliable information is usually obtained within a reasonable time in children under 7, but that for every year after 7 the difficulties rapidly increase. Given much time and skill these difficulties can sometimes be overcome, but it is very doubtful whether children of over 10 are suitable subjects for a really intensive retrospective study.

The Therapeutic Approach

Not only must we contend with the absence of witnesses and the forgetfulness of parents: suppressions and distortions of the truth, either conscious or unconscious, must be regarded as usual and inevitable. Such suppressions and distortions are only human when people feel themselves to blame, and it is obvious that the parents of children who are in trouble will tend to give a sugared picture of their influence on them. Though there is no complete solution to this problem, we know of one mode of approach more likely than any other to elicit the data we need—the therapeutic approach.

If people feel that the research worker is not merely an investigator but is also trying to help them, they will, as their confidence grows, reveal more and more of the truth, however discreditable and painful it may be. It is because mental-health workers have been prepared to help their patients that they have been relatively successful in elucidating problems of personal motivation and family dynamics. "No research without therapy" has become a slogan of the Tavistock Group, and by this is meant not only that it is unfair to human beings to subject them to an inquiry into the most intimate and perhaps most painful experiences of their lives without in return giving them such help as lies within our power, but that it is not possible to obtain accurate and meaningful research data unless we do so. An illustration may help.

A 6-year-old boy was brought to the clinic on account of bed-wetting, nervousness, and being generally miserable. The mother, who was intelligent and co-operative, was dealt with by a very experienced psychiatric social worker. The initial account of the family situation was that it had been a happy marriage and the baby was wanted. Not for twelve months did the mother acquire the courage to tell the true story of the boy's birth. He had been conceived before marriage, and as a result she had felt forced to marry a man whom she felt quite unsuitable. Had the psychiatric social worker not been experienced, patient, and helpful, the account of this boy's early social experiences would have been pitifully inaccurate and misleading.

The reliability of data is of course a major issue in all research, and in no field is it more difficult to ensure or to test than in the psychiatric. One check which should be used is the employment of two research teams trained together and working on identical lines and on similar samples, but independently. If there are no significant differences in their findings, there is a strong presumption that the findings of each are reliable; if there are significant differences, these can be measured.

Enough has been said to demonstrate that effective and reliable research work in this field can be done only by highly trained workers, familiar with both research and therapeutic techniques, and that the number of cases which can be dealt with by any one worker in the course of a year is necessarily small. This is a pity, but unless we face these realities squarely we shall merely waste our money and efforts on inadequate investigations, leading to ambiguous and erroneous conclusions.

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A study of the National Health Service, particularly its operation in the field of preventive medicine, forms part of the programme arranged by the British Council for Dr. Benjamin Viel, Professor of Hygiene and Preventive Medicine in the University of Chile, Santiago, who is spending two months in the United Kingdom under the council's auspices. Dr. Viel, who is 36, studied under a Rockefeller Fellowship at Harvard and Johns Hopkins Universities, qualifying as Master and Doctor in Public Health. He was appointed Professor of Epidemiology in the University of Chile in 1944 and two years later took up his present position. He has represented Chile at the Pan-American Public Health Congresses in Washington and Rio de Janeiro, and was awarded the "Corvalan Melgarejo" prize for the best work presented to the Sociedad Medica of Santiago in 1943.

CHILDHOOD SPEECH DISORDERS AND THE FAMILY DOCTOR

BY

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There are many aspects of speech and its disorders which are not fully understood. Although the literature is considerable it is not readily accessible to the busy practitioner, and the variations of expert nomenclature are a further hindrance to general interest and understanding. Mindful of this and of our own diagnostic uncertainties, we thought it would help the family doctor to share some points learnt from the treatment of 1,500 children in this region. The main work has been done by the speech therapist (M. M.), but recently we have studied together in more detail examples of the disorders met with in this series. Their percentage distribution was : stammering 40, dyslalia 26, cleft palate 16, severe or partial deafness 7, defective articulation without evident lesions of the nervous system 5, mental retardation 4, and cerebral palsy 2. This brings out the numerical importance of stammering and dyslalia but fails to reveal the important part played by mental deficiency in the delayed or defective development of speech. We shall not attempt a specialist account of these difficult problems but try to describe the main clinical features of the common disorders, their probable outcome, the degree of investigation possible in the family practice, and the best time to seek specialized help.

The development of speech in the normal child has been described by Dr. M. D. Sheridan in the *British Medical Journal* of May 19, 1945 (p. 707), and will not be repeated here. In fully developed speech there are three main phases: the reception of sounds by the ear and brain, the interpretation and regrouping of these sounds and their associated ideas, and the final expression of these symbolic sounds in speech. For the purpose of this paper we shall refer to these as the receptive, formative, and expressive aspects of speech and group our case histories under these headings. This mechanical simplification and separation of a finely integrated function cannot be pressed too far, as the frontiers between these three divisions are not always clearly defined. Nevertheless this anatomical concept of speech and its disorders is reflected in the clinician's approach to the affected child and justifies its use in an article of this kind.

Receptive Difficulties

Case 1.—This patient was the third child in a family without deafness or speech disorders. Pregnancy and birth were normal and she has had no serious illness. When first seen at 3 she appeared intelligent, but lacked understanding for speech and had made no attempt to speak. There had been some babbling sounds at one period, but these had not persisted. She ignored all the common household noises, such as opening doors, running taps, and the radio, and was in fact obviously deaf. She attended for treatment once every two months, and soon learnt to pick out familiar objects, when named, by watching the speaker's lips. Her mother was shown how to encourage the use of lip-reading at home; the result was good, so that at 5 years she was admitted to a school for the deaf, and had developed a considerable amount of understanding for speech. The hearing loss in this child was so severe that she was unable to appreciate the use of voice and had made no attempt to produce words herself.

Case 2.—An only child was first seen at the age of 6. There was no family history of deafness or defective speech. Born normally at term, she sat up at 6 months, walked at 17 months, and could feed herself by 18 months. She had measles at 1 year. She made no attempt to speak before 2, but after this speech developed slowly with silent intervals, and she could say "mummy" and "daddy" by 3 years. She always responded to household sounds, and the mother did not think she was deaf. She liked books and stories, but at 4 her mother noticed that the child watched her lips when she was reading to her. She played naturally, alone and with other children. At 5 her tonsils and adenoids were removed to improve her speech, though there was no history of tonsillitis, otitis, or nasal obstruction. When first seen speech was not intelligible, though she talked freely. She was unable to understand what was said if she could not see the speaker's face, though aware that someone was talking. Audiometric testing showed defective reception of the higher frequencies required for the hearing of speech, and treatment consisted in correction of the defective articulation followed by special help with reading. This child was reading well at 7½ years of age and was able, though not without difficulty, to maintain her place in a normal school.

The delayed development of speech in these children arises from a failure to understand speech owing to complete or partial deafness. In Case 1 there was no response, even to common household noises. Such a child would profit from a nursery school at the age of 3, and be ready for a school for the deaf at 5. Further, the mother can help the child to lip-read at home, and his progress at school will be more rapid if he has acquired some understanding of speech beforehand in this way.

Children with useful hearing for the lower frequencies of sound, but with deafness for the higher frequencies, are more difficult to detect. Like the child in Case 2, they can hear the passing car or the closing door and will listen to the radio and recognize and even imitate tunes correctly, but they show little interest in stories and conversation. The age at which speech will develop varies with the degree of hearing loss, but the common early picture is of an alert intelligent child without speech or understanding for speech, using gesture freely, and often as he grows older having tantrums when he fails to make himself understood. When speech does develop in these children the consonants mainly affected are "f," "s," "sh," and "th."

Accurate assessment of the hearing loss is necessary in planning for the future, and audiometry is helpful in this respect. In a young child with little or no understanding for speech this is not an easy matter, and several attempts may be necessary before a reliable result is obtained. However, it is usually possible to gain useful information when the child is 3 to 4 years or older, but if deafness is suspected the mother should be shown how to encourage lip-reading as early as possible. If good progress is made, education in the normal school may be possible, or a school for the partially deaf may be required.

Hearing-aids are not as a rule helpful, nor does a raised voice make speech more intelligible. Where the true nature of the defect has been missed and the children have remained in a normal school they tend to become worried and anxious, and have been classed and even certified as intellectually subnormal. If the hearing loss is not too severe most of these children eventually develop language and learn to express themselves in speech and writing. Middle-ear disease with partial deafness is rarely the cause of defective development of speech, and the removal of tonsils and adenoids is never the cure.

Expressive Difficulties

Case 3.—This patient was an only child aged 16. His mother died when he was 7, and at that time he could neither talk nor walk, was regarded as mentally defective, and was sent to a special institution. One of the nurses was interested in the boy and doubted the diagnosis of mental defect. She later married the boy's father and they decided to bring him home. He was then 14 years old and still unable to walk and talk. When first seen by us eighteen months later he could, through the efforts of his stepmother, walk, dress himself, talk, read, and had secured a job delivering newspapers. The condition was one of severe spastic diplegia with educational backwardness. Speech was clumsy but intelligible. When seen again three years later he was still delivering papers, attending evening classes, and making good progress in writing and composition. Speech and walking had continued to improve.

Case 4.—A girl, the second child of a family free from speech disorders, was born normally at term and weighed 6 lb. 6 oz. (2.89 kg.). Her condition in the early weeks of life gave rise to concern, and even after this period suckling was unsatisfactory. At 1 year her general development seemed normal and she could take solid food. She sat up at 10 months and walked at 18 months, but was "clumsy on her feet." She talked freely from 18 months onwards, but speech was not intelligible. She played normally, was fond of books and pictures, was not clumsy with her hands, and has done well at school. At 4 she had otorrhoea, followed by a thick nasal discharge intermittently for the next two years. The tonsils and adenoids were removed at 5, but there was no improvement in her speech. When first seen at 6 her speech had a nasal quality, and resembled that of a child with cleft palate. She made the correct tongue and lip movements for all consonants, but air pressure was inadequate for normal articulation, since the soft palate did not move effectively. The tongue was small, firm, rather thick, and did not protrude well. The nasopharyngeal sphincter was not efficient during speech, and there was occasional regurgitation on drinking. Examination revealed spastic diplegia involving the tongue and palate, with no obvious spasticity in the arms but with moderate spasticity of the legs and extensor plantar responses. Treatment included tongue and palate movements under relaxation, and blowing games to increase palate movement. Improvement was noticed within a few weeks, though speech deteriorated when she was nervous or excited. In this case the speech defect was more obvious than the spasticity of the legs, so that until 6 years of age the diagnosis of spastic diplegia had not been made.

We have used the term "expressive" to describe these children in whom the main difficulty is with articulation. In Cases 3 and 4 this failure or clumsy function of the speech musculature was associated with a more general spasticity arising from a probable failure of cerebral development. One danger for the practitioner in the more severely affected members of this group is to attribute the lack of speech, the sometimes rather vacant expression, and the emotional lability which may accompany cerebral palsy to mental defect. A careful examination of the nervous system, with special attention to the shape and mobility of the tongue, the tendon reflexes, the plantar responses, the presence of jerky or twisting movements of the arms, and the child's gait, should enable him to reach a correct diagnosis or at least a strong suspicion in most cases. Though walking and talking are usually late, the mother's account of the child's general behaviour will show that intelligence is good. Speech therapy is most effective in these cases when associated with physiotherapy based on relaxation and the development of muscle control. This can be started between 3 and 4 years of age.

Case 5.—A boy aged 4 years 8 months has one elder brother of 7 whose speech is normal. The pregnancy and delivery were satisfactory and the birth weight was 8 lb. (3.63 kg.). He was breast-fed and suckled well. He sat up at 9 months and could feed himself by 18 months. Play was normal from the beginning. He drew and painted, modelled with plasticine, and played well with other children. Speech started at 3 years, but was never intelligible. There was nothing on examination to suggest disease of the nervous system, and no evidence to indicate brain injury or cerebral maldevelopment. Hearing and understanding for speech were normal, and there was no evidence of intellectual defect. He could use the plosive consonants p, b, t, d, k, and g normally. The fricative sounds f, s, sh, and th could be made in isolation, but he could not associate them with a vowel sound at the beginning or end of a word. He had therefore substituted plosive sounds, and "fire" was "pire," "seesaw" was "teetaw," "jam" was "dam," and so forth. He substituted "w" for the sounds "y," "l," and "r," so that "lamb" was "wamb," "red" became "wed," and "yes" was "wet." Tongue movements were not well controlled in speech, though tongue movements not associated with speech appeared normal. Ability to co-ordinate the movements required for the combination of a fricative with a vowel sound developed slowly, and two years after the beginning of treatment speech was correct if the child spoke slowly. He is making good progress in school.

The precise cause of the defect in this last child is not clear, but the clumsy movement and poor control of some of the muscles of articulation would seem to be of organic origin, even though there are no other abnormal physical signs in the nervous system. In different patients in this group the faulty movements may affect mainly the lips, tongue, soft palate, pharynx, or larynx, but the incoordination is limited to its use in speech and is not obvious when the same muscles are used for other purposes. There may be difficulty in joining a consonant to a vowel, in the coordination of articulation and phonation. Speech may be good when slow, but the neuromuscular control inadequate for the rapid movements of normal speech. Treatment should be instituted about the age of 4 years, as there is often little spontaneous improvement, and even with therapy the defect may sometimes continue into adolescent or adult life.

Formative Difficulties

With the exception of the group of disorders illustrated by Case 5 there is a clear relation between structural defect and the receptive and expressive difficulties already described. The unravelling of formative difficulties is a more difficult problem. The simplest, and rarest, of these disorders is the aphasia which may accompany severe hemiplegia, whether congenital from birth injury or developmental abnormality, or acquired in the early years of life through encephalitis or vascular accident. In some of the children in this group the defective interpretation and regrouping of sounds and their associated ideas is further complicated by clumsy articulation. Speech also cannot be dissociated from general intelligence, and it is in children with formative difficulties that the need for excluding a wider mental deficiency most commonly arises.

This problem is well illustrated by Case 6. Although there may well be some general mental defect the specific failure in the boy's capacity to formulate language seems quite out of proportion to his general intellectual development, and apart from his defective speech his intelligence might well have been sufficient for effective education in the ordinary school which he attends. In Case 7, on the other hand, the question of mental or neuromuscular defect does not arise, but the language difficulty remains.

Case 6.—A boy aged 12 is the fourth child. The pregnancy and delivery were normal. He walked at 11 months and was feeding himself at 18 months. In spite of normal development in other ways he did not begin to speak until 5. He seemed to have considerable understanding for speech, and used noises or gestures to attract attention. When first seen by one of us (M. M.) at 7 his speech was unintelligible, though all consonants could be articulated normally in isolation and initially

in simple words. He did not use any final consonants and omitted medial ones. Attendance for treatment was erratic and finally lapsed. When seen again five years later there was very little improvement in speech, and the defective articulation was unchanged. He could not read or recognize letters, though he could write his name in large script. He brought home library books and newspapers and asked his mother to read them to him. He played normally with other boys and was capable of looking after himself and leading an independent life. Yet on a series of intelligence tests, including the Terman–Merrill, Drever Collins, and Alexander performance tests, he scored between 60 and 65%. A fair assessment of his innate intelligence, however, is difficult to obtain. He is now educationally retarded, and to this the severe speech defect must have contributed.

Case 7.—A boy aged 4 years 6 months, the second child of intelligent parents, was born normally at full term and weighed 7 lb. (3.18 kg.). Progress was satisfactory until a severe attack of gastro-enteritis at 8 months. He subsequently sat up at 1 year and walked at 20 months. He began to say a few isolated words at the same time, but progress in formulating language has been slow. When examined at 4½ he was well developed, with no abnormality in the nervous system. He was not deaf, and his intelligence quotient with the Merrill–Palmer performance test was 102. Though speech was unintelligible all consonants were used correctly in isolated words of one syllable. In sentences many words and syllables were omitted. Nouns predominated in his speech, but words were not used in correct sequence. He could recite nursery rhymes, again with omissions and inversions, but such practised word sequences were more correct than spontaneous speech. Memory for events was good, and he recognized tunes on the radio, though he could not imitate them accurately.

Degrees of failure to develop the use of spoken language vary from the child who is only a little late in speaking to one with a defect so severe that education in the normal school is impossible. When speech does eventually develop, articulation may be slightly or seriously defective, and in severe instances this may persist into adult life. Such severe cases will require education in a special school for speech disorders, of which at present there is only one in this country

Dyslalia

Cases 8 and 9 illustrate a common speech disorder for which we reserve the term dyslalia. It is not easy to understand the nature of their defect—a defect which can be severe yet resolve spontaneously, and is remedied with relative ease. A possible explanation is that during the early development of speech the rapid development of language is associated with a partial failure of both perception and imitation of speech sounds. This leads to faulty habits of articulation, which persist when the underlying dysfunction has passed. We might compare it to the early stages of learning a foreign language when vocabulary often outstrips accuracy in pronunciation. Such children are unaware that their speech differs from that of other people, and they become distressed when they cannot make themselves understood. Because we feel that the articulatory defects shown by these children could arise from such a primary lack of fine discrimination between speech sounds we are inclined to leave them for the present in the "formative" group of disorders.

Case 8.—A girl aged 9 had developed normally except for speech which, though starting early, was unintelligible. She was worried because she could not talk like other girls and was anxious to speak correctly. She could read freely but not intelligibly, and was holding her own at school in spite of her disability. Her speech showed many consonant substitutions—for example, "g" = "d" ("gate" = "date"), "f" = "t" ("fat" = "tat"), "s" = "t" ("see" = "tee"), "k" = "t" ("coat" = "tote"), and "w," "y," "l," "th," and "r" were not used in speech. The tongue and palate movements were normal, and there was no abnormality in the nervous system. She attended for treatment once a week, and within three months all consonants were normal and could be used correctly in reading, though not in conversation. After a further five visits during the following two months speech, as well as reading, was normal.

Case 9.—A girl aged 5 developed normally apart from her speech. There is no family history of defective speech or deafness. At 4 her tonsils and adenoids had been removed to improve her speech, but at 5 this was still unintelligible. She tried to speak early, but had recently become conscious of her inability to make anyone, even her mother, understand her, and had stopped trying. She played mostly with her sisters, as other children teased her. She had reacted to this by nipping them, and there had been complaints from their parents. The mother said that the child would sing to herself when she went to bed at night, and appeared to be making a song of the doings of the day. There was no developmental delay in any other respect, and her intelligence was considered above average by her teachers. After treatment on five occasions the mother said the child was less worried and was beginning to talk again, though not intelligibly. At the end of four months speech was making good progress, and three months later she was discharged.

The difficulty in these children varies from the defective articulation of one consonant to multiple substitutions and omissions, with varying degrees of intelligibility. Unlike the previous types of structurally determined speech defect, articulation often improves quickly and spontaneously in these children, although the defect may persist into the early years of school life, proving a serious handicap and resulting in much unhappiness from mocking and teasing by other children.

In all cases of this type speech eventually becomes normal, and in the majority without treatment. Treatment between 4 and 5 years of age, however, is useful in allaying the mother's anxiety, rescuing the child from nagging correction at home, and allowing him to start school without the handicap of defective speech. It has been our practice to see the mother and child once a week. The mother is present during treatment and is shown how to carry out simple speech exercises. With guidance the mother is often the one who can best carry out practice at home under easy and natural conditions. The exercises are practised for not more than five to fifteen minutes a day, depending on the age of the child, and include rhymes and the repetition of stories. During this period the mother is asked to see that the child's actual conversation is not corrected until he is able to use all consonant sounds easily in exercises and rhymes. By this time such correction is usually unnecessary, as normal articulation is being gradually and spontaneously incorporated into speech and self-consciousness about speaking thereby avoided.

Cleft Palate

A detailed consideration of the speech disorders associated with cleft palate is out of place here. It would be fair to say, however, that where the palate has been successfully repaired before speech is established, speech therapy is rarely required. Case 10 emphasizes the need for full closure of the cleft and the value of speech therapy in those children in whom operation has been delayed or is only a partial success.

Case 10.—A boy aged 3 was born with a bilateral complete alveolar cleft of the palate and lip. The lip was operated on at 3 months and the palate at 27 months. He is an intelligent child, and was talking a great deal before the second operation. When first seen he had a small hole in the hard palate, but speech therapy was started without a dental plate. He could not articulate any consonants except the nasal ones, "m,"

"n," and "ng." There was nasal escape of air on blowing. Speech therapy continued for three months, but the child was obviously finding it difficult, and a small dental plate was made to cover the aperture in the hard palate. Subsequently blowing was normal without a nasal leak. Speech then developed easily and quickly, and was normal within a year. It is probable that, if such a dental plate had been provided within a few months after operation, normal speech might have developed spontaneously and without the necessity for speech therapy.

Stammering

In view of its frequency no account of the common speech disorders in childhood would be complete without reference to this distressing condition. Stammering occasionally begins when the child starts to speak, but it is more usual at the age of 3 or 4, after a short period of normal speech. In many such cases the stammer clears up rapidly, and transient difficulty of this kind is so common that, like transient stammering at moments of emotional stress in adult life, it may be considered within the range of normal development. The most frequent time of onset, however, is during the first year of school life, and such a stammer may persist, especially where the child is unwisely corrected by an over-anxious parent.

Against a purely mechanical explanation of stammering are the fact that the child possesses the physical mechanisms for perfect speech and can in most cases use them in singing, and in reading or reciting when alone, that the only physical signs on examination are those of anxiety often associated with tics, and that disturbing situations strongly influence the severity of the stammer just as they may provoke a stammer in an adult who has been free since early childhood. Stammering is nearly always worse on interviewing an elder or superior, and it is much commoner in boys than in girls. A family history is common, but convincing statistical evidence of a specific relation to left-handedness is lacking in our patients.

The vexed issue is whether persistent stammering is a psychoneurotic symptom based on persisting emotional conflict and requiring psychotherapy, or whether the speech disorder often outlasts its primary cause and needs mainly symptomatic treatment in the form of speech therapy. Our own attitude is that, while in every case the family situation should be considered and the child's temperamental difficulties appreciated and assessed, most stammering children can be adequately treated by a speech therapist, supported in the more severe and intransigent cases by an experienced psychiatrist or physician.

The practical point in management is that as in other similar disorders fear of failure and ridicule are potent factors in the maintenance of stammering. In the case of the early stammer in a young child it is mainly the mother who needs reassurance and guidance in handling the situation. If this is successful she will be willing to leave the child alone so that the symptom ceases to be a source of secondary anxiety. If, however, the condition has persisted for as much as six months with no signs of improvement, group treatment may help, and if given at 5 or 6 years of age it will often assist the child to regain normal speech. When treatment has been long postponed faulty habits of speech become more firmly established, and improvement becomes progressively more difficult to achieve.

Speech Disorders in Mentally Retarded Children

Many children who first come to the family doctor with delayed or defective speech will prove on closer scutiny to be mentally defective. Idiots never develop intelligent speech, or remain limited to a few isolated words which first appear in later childhood. Imbeciles, as, for example, the familiar mongol, rarely speak before 3 and cannot master short sentences until 6 or more years of age. As we move upward through varying degrees of feeblemindedness into the borderland of normality, so the delay and disorder in the development of speech diminishes, and superficial contact with some feebleminded children may suggest that speech is normal. Careful inquiry and repeated observation will show that in most of these the onset of speech is delayed, the vocabulary limited, and articulation often clumsy even after schooling has started.

It is necessary, however, to labour the important if obvious fact that, though mental defect is often associated with defective speech, defective speech sometimes (as in Case 3) leads to the erroneous diagnosis of mental defect. The assessment of the child's general development is therefore of particular importance in all children with speech disorders. Even in the presence of impaired speech a simple inquiry for the "milestones"—the age of sitting up, walking, feeding himself, becoming clean and dry, dressing himself, ability to play alone and the type of play at different ages—will often enable a provisional assessment to be reached. It is usually wise for both the doctor and the parents to "take time to pause" before accepting a diagnosis of mental defect. Observation of the child for a year or more may be necessary in the less severe cases and will help the parents to reach the diagnosis for themselves.

Once mental defect is consistently suspected expert confirmation or investigation is required. The practitioner will generally lack the time and training necessary for the accurate measurement of intelligence which presents special difficulties in children with impaired speech. Yet the experienced paediatrician or psychiatrist, supported where necessary by a variety of performance tests, can reach an accurate assessment in most cases. When this has been done speech therapy may sometimes play a useful if limited part in the education of the child, since many of the associated speech disorders are similar to those found in mentally normal children.

The Investigation of a Child with Defective Speech

From the histories and comments given it will be seen that speech disorders fall into a number of broad categories which the practitioner can recognize by the familiar techniques of clinical medicine. When faced with the individual patient five main questions have to be answered. Is he severely or partially deaf? Is his speech disorder part of a more general disturbance of neuromuscular function? Has the child the formative speech difficulty we have called dyslalia? Does he stammer? Is he otherwise mentally normal?

In seeking an answer to these questions attentive listening to the mother's account of the child's development from birth is without doubt the most helpful procedure. Failure to respond to all kinds of noise will suggest deafness. An interest in sounds or music but not in speech, stories, or conversation, and with delayed development of understanding for and the use of speech, suggests high frequency deafness. Delay or clumsiness in suckling, walking, and talking will call for a careful examination of the nervous system. Where everything is normal apart from speech, listening to the child recite a rhyme will bring out the substitution defects of dyslalia or perhaps a stammer.

In those children with clumsy speech and no evident lesion in the nervous system a careful history may reveal an early period of dysphagia or muscular spasticity which has now been overcome. In examination, too, it is important to watch the control and range of tongue movement,

especially the child's capacity to point the tip and raise it over the upper lip towards the nose. With very rare exceptions tongue-tie is not a cause of speech defects and snipping the fraenum is a useless and sometimes dangerous operation. The establishment of mental deficiency calls for repeated and usually expert observation and has already been considered.

Conclusion

Speech disorders are common in childhood and often cause considerable distress to both the child and his parents. Although the speech mechanism is not fully understood, its disorders can be tentatively placed in a limited number of broad groups which can be differentiated in the majority of cases by a careful history of the child's development and simple physical examination.

The accurate assessment of intelligence and hearing, and the treatment of the more severe disorders, will call for expert help, but a provisional diagnosis will often enable the practitioner to inform and reassure the parents, and to see that the child reaches the speech therapist at the optimum time.

ACUTE LARYNGO-TRACHEO-BRONCHITIS

BY

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The following case is worthy of record in view of its relative rarity, the difficulty of treatment, and the rapidity of the clinical course.

Case Report

A medical officer from an outlying unit telephoned requesting the admission of a child aged 18 months, son of a soldier, as a case of treatment-resisting bronchitis, stressing that he was not acutely ill. The child was admitted two hours later.

He had had measles three weeks previously, and since then had a loose cough with some anorexia and loss of weight. There had been one previous attack of bronchitis, at the age of 6 months, and he had been immunized against diphtheria. There was nothing relevant in the family history. He was brought into the hospital by his father, who stated that the child was quite happy.

On examination the patient was not ill. Laryngitis was noticeable when he cried. The temperature per rectum was 99.2° F. (37.3° C.), pulse 104, and respirations 24. The ears, heart, abdomen, and C.N.S. were all normal. There was much mucopus in the nose; the tonsils were large, but looked fairly healthy; and pharyngitis was present, with a post-nasal drip. The tonsillar glands were palpable. Lungs: The percussion note was hyperresonant, and scattered high-pitched sibilant rhonchi were heard throughout both lungs during expiration. Bronchial breathing was heard over the spinous processes down to and including the fourth thoracic vertebra. An *x*-ray film of the chest was normal.

This was considered to be a case of asthmatic bronchitis with laryngitis; the child was therefore given "argyrol" and ephedrine nose drops and a stramonium and iodide mixture. Steam inhalations were not thought necessary, as the laryngitis was not severe.

At 3.30 p.m., three hours after admission, he suddenly became cyanosed and was pitifully frightened. On examination he had an obvious laryngeal obstruction, there was indrawing of the lower ribs on inspiration, the accessory muscles of respiration were in play, and large numbers of high-pitched sibilant rhonchi were heard throughout the lungs during expiration. Subcutaneous adrenaline gave marked relief and he was put in an oxygen tent. A diagnosis of laryngeal diphtheria was made, and he was given 30,000 units of antidiphtheria serum intravenously into the external jugular vein and 200,000 units of penicillin intramuscularly six-hourly. The ear, nose, and throat surgeon was consulted, and it was agreed to perform a tracheotomy. Laryngoscopy revealed no diphtheritic membrane, and a swab was taken of the oedematous and hyperaemic larynx. A tracheal swab was also taken when the tracheotomy tube was established.

The child spent a restless night in the oxygen tent after an initial improvement from the tracheotomy which lasted only about two hours. The next morning the respirations were extremely embarrassed and he was obviously dying. On examination fine sticky crepitations were heard in both lungs and the right base showed signs of consolidation, with an impaired percussion note and diminished air entry. As the child was vomiting, sulphadiazine, 0.5 g. four-hourly, was given intramuscularly. His condition deteriorated, and he died 30 hours after admission. Laryngeal and tracheal swabs gave a pure growth of coagulase-positive, penicillin-resistant *Staph. aureus* but no evidence of Klebs–Loeffler bacillus.

Post-mortem Findings.—The tracheotomy airway appeared clear; the larynx, trachea, and bronchi were grossly inflamed; and the lumina were filled with pus. Bronchopneumonia with areas of lobar consolidation was found in the right lung, with interstitial emphysema. The causative organism seems to have been *Staph. aureus,* which was isolated in pure culture from the patient before death and from the lungs post mortem. Death was due to bronchopneumonia following a fulminating laryngo-tracheo-bronchitis.

Histological Report.—In the base of the right lung there were obvious areas of atelectasis. Some of the bronchioles were blocked by inflammatory exudate. Only slight inflammatory changes were present in the adjacent alveoli. This picture was true of the left base and both apices. In the trachea and larynx there was almost complete replacement of the epithelial lining by an acute inflammatory exudate which extended to the submucous layer. Nothing abnormal was noted in other tissues. The picture was indicative of an acute laryngo-tracheo-bronchitis with atelectasis. Actual pneumonic changes were minimal.

Discussion

The signs, symptoms, and post-mortem findings make this in all respects a typical case when compared with the picture presented by Chevalier Jackson (1946) and Bonham-Carter (1949). There is one exception—namely, the clinical and post-mortem evidence of bronchial spasm.

Since this case, two others have been seen, one of which went into acute laryngo-tracheo-bronchitis straight from an attack of asthma. The other was identical with the case reported.

Banks (1949) stated that in his experience penicillin and sulphonamide therapy had no effect in this disease, suggesting that it may have a virus aetiology. In view of this statement, the experience with the above case, and the reports of penicillin-resistant *Staph. aureus* infections (Barber *et al.*, 1948, 1949), the second case was treated with streptomycin, with dramatic, rapid, and complete recovery. Penicillin and sulphonamide compounds were not given at all.

Unfortunately streptomycin, with its unwelcome side-effects, is not the ideal weapon. In view of the incidence of penicillin-resistant *Staph. aureus* infections in this department of the hospital, "aureomycin" was obtained for future use. A leading article in the *British Medical Journal* (1949) stressed the value of this antibiotic in penicillin-resistant *Staph. aureus* infections and the absence, so far as is known, of toxic effects.

The third case of acute laryngo-tracheo-bronchitis, in a 3-year-old child, was treated with aureomycin in 50-mg doses six-hourly for six days. Once again recovery was dramatic, rapid, and complete. A similar result occurred in three cases of bronchopneumonia which were resisting penicillin therapy and then received aureomycin.

Moncrieff and Weller (1949) in a preliminary communication have given encouraging reports on the use of chloramphenicol in this disease.

It therefore seems that we are at the moment up against a disease which, be it virus in origin or not, kills by virtue of penicillin- and sulphonamide-resistant bacteria, and that the newer antibiotics may be the weapon for which we are seeking to combat it. This is of course true, as has been briefly mentioned, in cases of penicillin-resistant bronchopneumonia and bronchitis; but acute laryngo-tracheobronchitis has been stressed here because of the extreme rapidity of its course, and because if the patient is not treated promptly and energetically with a suitable antibiotic no second chance remains.

Summary

A case of acute laryngo-tracheo-bronchitis is described in its clinical course and pathology.

Treatment is discussed in view of the increasing incidence of penicillin-resistant bacteria.

It is stressed that, owing to the rapid and fatal course, the first line of treatment must be with one of the newer antibiotics.

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X-RAY EXAMINATION IN "PNEUMONIA"

BY

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Various opinions have been expressed on the advisability of radiographing cases of "pneumonia." Some people advocate *x*-raying every case; at the other extreme are those who believe that such a course is unnecessary and that in general practice the need arises only occasionally. This article has been written with the object of trying to assess the usefulness of radiography in cases of "pneumonia."

The term "pneumonia" has been used to describe a symptom-complex affecting the respiratory system. It consists of several, and sometimes all, of the following symptoms: malaise, fever, cough, pain in the chest, sputum, dyspnoea, and haemoptysis of small amount. The general picture of every patient in this series has been such that during the acute stage of the illness a diagnosis of pneumonia of bacterial origin, atypical pneumonia, or pneumonitis might have been justified from a consideration of the symptoms and physical signs alone. The diagnosis in these terms was made either by the general practitioner referring the patient or by the writer. Cases with the syndromes designated by the terms influenza, dry pleurisy, and pleurodynia have been excluded. Cases sent up correctly diagnosed as something other than "pneumonia"—e.g., pleural effusion, spontaneous pneumothorax, neoplasm, etc.—have been excluded whatever their symptoms. The cases discussed were consecutive, and were selected because they showed an abnormality on radiography. All were followed up until a normal skiagram was obtained or a clear diagnosis of disease arrived at. With these criteria 228 cases, referred during 18 months, were selected.

The patients were all referred to, or a second opinion on them was sought from, this chest clinic, which is situated in a county town of about 60,000 people. The surrounding countryside and small towns augment the total number covered to over 140,000. The series is in no way unusual unless it be exceptional for a large percentage of the surrounding practitioners to be intent on diagnosing pulmonary tuberculosis early, and to be aware that this is better done by radiography than by the stethoscope.

Classification of Cases

The cases can be divided into three groups in accordance with when they were first seen. (1) Those referred during an acute illness suggestive of pneumonia, often after no response to the sulphonamides and penicillin. (2) Those referred specifically as having recently had pneumonia and as having failed to resolve normally, or because the practitioner wished to be assured of normality. (3) Those referred for an opinion as being suspected cases of pulmonary tuberculosis after an acute illness suggestive of pneumonia but variously diagnosed.

Table I shows the number in each group, and divides them into the age groups 0–14 and 15 upwards.

TABLE I

Age	Group 1	Group 2	Group 3	Total
0–14 ..	1	19	28	48
15+ ..	37	93	50	180
Total ..	38	112	78	228

In Group 1 there were 11 cases which resolved normally and 27 which either presented complications or were not pneumonia. In Group 2 the corresponding figures were 61 resolving normally and 51 others. In Group 3 the figures were 55 resolving normally and 23 others.

In Table II the abnormalities apparent on radiography are listed, and the number occurring in each group is shown.

TABLE II

Abnormality Found	Group 1	Group 2	Group 3	Total
Pulmonary tuberculosis	9	20	3	32
Delayed resolution	4	12	13	29
Empyema	4	2	1	7
Sterile serous effusion (non-tuberculous)	2	2	1	5
Tuberculous effusion	1	4	1	6
Bronchiectasis	2	8	1	11
New growth	2	1	1	4
Lung abscess	1	0	0	1
Spontaneous pneumothorax ..	1	0	0	1
Cardiac	0	2	1	3
Cough fracture	0	0	1	1
Death (post-operative pneumonia)	1	0	0	1
Total	27	51	23	101

Out of the 228 cases referred with the symptom-complex of "pneumonia," 127 resolved normally and were classed by the writer as pneumonia, atypical pneumonia, or pneumonitis. Out of the other 101 cases one, a patient with post-operative pneumonia, died; 29 ultimately resolved completely, but with varying periods of delay, and 12 others suffered direct complications of their pneumonia. In a further 19 a second condition as well as pneumonia was present, being brought to light by radiological examination. This group included three cases of known phthisis.

Finally, 40 cases were judged to have diseases other than pneumonia. These conditions will be briefly analysed under appropriate headings.

Complications of Pneumonia

Delayed Resolution.—This term has been used when abnormal shadowing, ultimately disappearing completely, was still present at least three weeks after the first radiograph was taken. In practice, as the radiograph was usually taken about 7 to 14 days after the beginning of the illness the period was often longer than three weeks. Twenty-nine (about 15.5%) fell into this category. This high figure cannot be compared with any obtained from an average series of pneumonias, as many cases were referred because the illness was in some measure atypical. It has been affirmed by Davies *et al.* (1935) and Morgan and Wylie-Smith (1943) that delayed resolution is commoner in the older age groups. Their belief was not sustained by this investigation, in which four were below the age of 15, eight above 40, and the remaining 17 spread evenly between these ages. Table III presents these figures in more detail, showing the number in each age group and the percentage affected by this complication.

TABLE III

Age	No. with Pneumonia	No. with Delayed Resolution	Percentage with Delayed Resolution
0–14	47	4	8·5
15–40	84	17	20
41+	57	8	14
Total ..	188	29	15·5

In the majority of these cases there were no symptoms referable to the delay in resolution. A few complained of slight shortness of breath on exertion and the slow return of strength and energy. Abnormal physical signs had usually disappeared, although there was still radiological evidence of disease. In three cases, however, rales persisted over the area for several months after the *x*-ray film had returned to normal. In two of these, bronchograms showed no evidence of dilatation of the bronchi. In a further two patients evidence suggesting pleural adhesions remained after the lung parenchyma had apparently returned to normal.

Empyema.—Seven cases of empyema occurred—one in a child of 12 months. Four were cured by the aspiration of purulent fluid and the instillation of penicillin into the pleural cavity. One required rib resection and drainage. Diagnosis was delayed until seven weeks after the onset of the illness in this case, and communication had by this time been established with a bronchus. The seventh showed evidence suggesting a small pleural effusion from which needling withdrew no fluid. Purulent sputum occurred later in the illness and was seen by bronchoscopy to be coming from the lower lobe bronchus. This case was possibly a lung abscess; the weight of evidence suggested an empyema.

Sterile Serous Effusion.—The five cases had all received treatment with one or other of the sulphonamides. Penicillin had also been given in two cases. The history of the illness suggested pneumonia in every case, and blood-stained sputum had been noted in the four adult patients but not in the one child. Specimens of fluid were obtained from four cases, and were in each instance sterile and straw-coloured. The fluid was clear in one and opalescent in three. All eventually resolved without leaving any evidence of disease. Three effusions were small and two of moderate size. From one case 30 oz. (850 ml.) of fluid was withdrawn. This condition is quite common, and may be followed by the development of empyema.

Pulmonary Tuberculosis

Pulmonary tuberculosis was present in 32 cases. Three had been known to be suffering from this condition—one with positive and two with negative sputum. All three resolved normally, and were referred for fear of reactivation. Of the remaining 29 cases, four proved to have inactive and one active disease associated with pneumonia. Out of eight patients, therefore, only one was judged to be reactivated by the inflammatory complication, which occurred in two lobes affected by the proliferative form of tuberculosis. Baum and Amberson (1942) found pneumonia not uncommon in patients with pulmonary tuberculosis. Spread or reactivation of disease was likely only if the pneumonia was accompanied by suppurative disease or the exudative type of phthisis was present. Baum and Baum (1949) in a further investigation confirmed these findings and noted that reactivation was most probable in fibro-caseous tuberculosis and where the pneumonic process attacked a lobe containing such disease. Three of the above eight cases suffered from pneumonia of a lobe in which there was obvious radiological evidence of phthisis. Only one of these was of fibro-caseous type, and in this there was no change in the status of the primary condition.

Twenty-five cases were suffering from active pulmonary tuberculosis, including the one referred to above. Their symptoms were judged as being due to this condition in 24. In 21 of these the sputum was positive for tubercle bacilli. The varied nature of the symptoms of onset of phthisis is well known. Its insidiousness has been stressed to the depreciation of the more acute forms of onset. Rist and Ameuille (1922), Rist (1929), and Douglas, Pinner, and Wolepor (1929) have all drawn attention to the fact that many cases present with symptoms suggesting a pneumonic or influenzal process. They state that about 50% of cases are suffering from a pneumonic process of lobar or segmental distribution at the time of their first symptoms. About 25% present with symptoms of "pneumonia" and 25% with those of "influenza." More recently the same facts have been commented on by Toussaint and Pritchard (1944). Further support, but from a different angle, is given by Williams and Hill (1930). They analysed the diagnosis other than the correct one given to 1,199 patients suffering from pulmonary tuberculosis. Bronchopneumonia (12), pneumonia (56), pleurisy (165), and congestion of the lung (10) accounted for a total of 243 misdiagnosed cases. Thus nearly 25% had ostensibly suffered from symptoms suggesting the symptom-complex under discussion.

I am not of the opinion that the initial lesion in pulmonary tuberculosis is a pneumonic process of segmental or even broader distribution: the first symptoms of disease noticed by the patient are much more often those of acute illness than is generally appreciated. The insidious symptoms of cough, lassitude, and loss of weight will often on careful questioning be found to have been preceded by an acute episode. It has been said that phthisis progresses by means of a succession of pneumonias.

The symptom-complex that we are considering may be caused in a number of ways by pulmonary tuberculosis. In the 25 active cases found in this series an attempt has been made to decide what tubercu'ous process instigated the attack of seeming pneumonia. Twelve cases were associated with recent consolidation, either lobar or segmental

in extent, using the term "segmental" to describe one of the main broncho-pulmonary segments of less than lobar size. Eight were associated with recent spread, bronchopneumonic in type, in which evidence suggesting an older lesion was present. Two occurred at the time of collapse of a lobe. One was a case of typical acute tuberculous lobar pneumonia. One was brought to notice by an effusion complicating bilateral parenchymatous disease. One was a case of bilateral proliferative disease, which was probably reactivated by an atypical pneumonia, though it might possibly have been active and without symptoms previously.

Among the active cases there were five women, all under the age of 40, and 20 men, of whom 12 were over the age of 50. The age of the men should be specially noted, because there is a fairly widespread belief that phthisis with acute symptoms is uncommon in the older age groups. The single case of tuberculous pneumonia was in the youngest, a female aged 19. Of all the 48 children below the age of 15 none proved to have pulmonary tuberculosis, although it was frequently suspected by the medical attendant. This was to be expected, as primary tuberculosis, the only common form of the disease found in children, rarely presents with the symptoms under review.

Bronchiectasis

The 11 cases of bronchiectasis were found after the pneumonic process might be considered to have subsided. Eight were confirmed by bronchography. The other three cases presented radiographs typical of bronchiectasis six months or more after the acute attack. All had histories of past pneumonia and symptoms characteristic of bronchiectasis extending over many years. All were accompanied by abnormal physical signs.

Tuberculous Pleural Effusion

Six cases of pleural effusion, presumed to be tuberculous in origin, are included. None of the specimens taken from these cases disclosed tubercle bacilli either on direct examination or on culture. No special procedures such as described by Eberle (1949) were utilized to isolate tubercle bacilli. All the effusions were straw-coloured; four were clear and two opalescent. Nairn (1949) states that, apart from the demonstration of tubercle bacilli, it is impossible to decide the origin of a pleural effusion by laboratory methods. These cases were divided from the serous effusions of pneumonic origin on clinical grounds, special attention being paid to the history of onset of the illness, the presence of blood-stained sputum during the acute stage, and the response to sulphonamides.

Miscellaneous

New Growth.—In two of the four cases sudden collapse of an upper lobe was diagnosed as pneumonia. In one a pleural effusion was similarly mistaken, and the fourth presented as an abscess of the right upper lobe. The diagnosis was confirmed microscopically in each case, two specimens being obtained by biopsy during bronchoscopy and two at necropsy. All were dead within six months of their first attendance.

Lung Abscess.—No cause was found in the one case. It cleared quickly with penicillin and postural drainage.

Spontaneous Pneumothorax.—An incomplete pneumothorax—i.e., with adhesions—accompanying bronchitis and emphysema was found in one individual.

Cardiac.—Of the three cases one was suffering from a pulmonary infarct and auricular fibrillation, and one from pulmonary oedema and congestive failure which had been preceded by pyrexia of unknown origin. The third was a case of pulmonary oedema accompanied by left ventricular failure and hyperpiesia.

Cough Fracture.—One case of cough fracture of the sixth right rib occurred in a woman six months pregnant who also had an atypical pneumonia of the left lower lobe. Attention has been drawn to this relatively common condition by Cohen (1949). No proved case of cough fracture on the right side has been reported previously in pregnancy. Furthermore, the condition has hitherto occurred during the last three months only (Pauley *et al.*, 1949).

Discussion

From this short analysis of 228 cases of "pneumonia" there would seem to be little doubt about the value of radiography as an extra aid to diagnosis. It is wiser to err on the side of safety by taking a radiograph which shows no abnormality than to procrastinate with the risk of disease progressing.

The figures justify the use of radiography in "pneumonia" if only as a method of diagnosing phthisis. This disease, so amenable to modern treatment in the early stages and so difficult to arrest when advanced, is commonly thought to have most frequently an insidious onset. That this opinion is erroneous cannot be too often repeated. Twenty-nine new cases were discovered. Twenty-five were active, and 21 of these had sputum which was positive on examination for tubercle bacilli at the time of their first radiograph. Thus 11% of the total proved to have active pulmonary tuberculosis. On excluding these 228 from the new cases referred to the chest clinic over this period it was found that all other methods of case-finding produced only seven cases of active reinfection tuberculosis per 100 new individuals examined. The two groups are not strictly similar, but the conditions favour the second group, which contains new cases referred already diagnosed and the positive results of serial contact examination.

The percentage of abnormality in this series is high, and it should be clearly understood that if every case presenting with the pneumonia symptom-complex was referred many more radiographs with normal shadows would be taken. In point of fact, 27 cases (21 adults and 6 children) were seen during this period complaining of the necessary symptoms, but their films showed no abnormality. However, if we are to strive for increased accuracy in diagnosis, with the object of recognizing disease in its earliest and hence most curable stage, the idea that every radiograph taken must show an abnormality should be abandoned. Where chest conditions are concerned this concept has already gained much ground, furthered by the success of mass radiography. Now that an attachment for photo-fluorography can be employed with any efficient modern radiographic unit at the cost of a few moments' adjustment, the problem of the time and expense of taking full-size films, except when a lesion is revealed, has been satisfactorily solved. It is to be hoped that most areas will in the future be able to take large numbers of miniature films. Until such facilities are universal the best possible use must be made of existing apparatus.

Summary

Analysis has been made of 228 consecutive cases referred with the symptom-complex of "pneumonia."

Forty-one patients were suffering from complications of pneumonia; they included 29 cases of delayed resolution, 7 of empyema, and 5 of sterile serous effusion.

Twenty-five active and 4 inactive cases of pulmonary tuberculosis were disclosed.

Twenty-seven further cases of disease were revealed. They included tuberculous effusion (6), bronchiectasis (11), new growth (4), lung abscess (1), spontaneous pneumothorax (1), cardiac (3), and cough fracture (1).

It has been argued that radiography should be widely applied to the symptom-complex of "pneumonia."

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ADRENAL CORTICAL TUMOURS OF UNUSUALLY LARGE SIZE

BY

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Tumours arising from the adrenal glands are rare, especially those primarily in the cortex. It is well known that cortical hyperplasia of either a focal or a generalized type occurs during middle life and is often associated with feminine pseudo-hermaphroditism (Glynn, 1912). Where the hypertrophy is generalized, a gland may enlarge and even approximate in size to the kidney. All gradations occur between simple hypertrophy and true adenoma formation, and as the two conditions appear during the same age period it is sometimes very difficult to differentiate one process from the other.

Primary malignant tumours arising from the glandular elements of the cortex are usually referred to as adenocarcinomata—a term which has largely replaced that of hypernephroma. As it was common to misapply the older term to renal tumours and non-cortical adrenal tumours it is obviously an advantage to abandon it, but since the adrenal cortex is developed from mesoderm it is clearly not entirely accurate to employ a title which indicates an epithelial origin. However, most pathologists agree that the histological appearances are of "adenocarcinomatous" type, and I shall use the term here.

In the same way as difficulty is encountered in drawing a sharp line between simple hyperplasia and true adenoma formation, so it is not always easy to differentiate histologically between benign and malignant conditions. Thus when metastases can be demonstrated the diagnosis is clear despite the fact that the primary tumour may be made up of cells apparently identical with those of a normal adrenal gland. Often, however, spread occurs late, and large tumours are encountered which undergo necrosis and extensive haemorrhage yet possess thick well-defined capsules.

Adrenal cortical tumours may be further subdivided into those which are associated with hormonal disturbances and those which are not. Examples of cases occurring without hormonal disturbances are well recognized but rare. The majority occur in adults over the age of 30 and have been described in both sexes. Cahill *et al.* (1942) found five such cases in a series of 13 adrenal cortical tumours. The histological appearances vary from those that are well differentiated to those showing an extreme degree of anaplasia.

The following two cases fall into the group of tumours of adrenal cortical origin without hormonal disturbances. In each case the clinical picture was that of a large abdominal mass causing minimal discomfort. In both cases the tumours were discovered on routine examination, when the patient had sought advice for other symptoms. Both were easily removable at operation.

Case 1

A man aged 69, under the care of Sir Stanford Cade, was first seen at Westminster Hospital on May 28, 1949, complaining of difficulty in micturition. Five months previously he had noticed a painless swelling in the right side of the abdomen.

Examination revealed a heavily built man with dyspnoea on exertion and a blood pressure of 240/120, but without clinical evidence of Cushing's syndrome. Prostatic enlargement was thought to account for his urinary symptoms. A very large, tense, immobile mass was present in the right side of his abdomen extending up to the xiphisternum and across the midline towards the left. The possibility of a large hydronephrosis was excluded because the excretion pyelograms showed normal kidney patterns.

At laparotomy on June 7 a large retroperitoneal tumour lying anterior to and above the right kidney was discovered. No adrenal gland could be found on that side. The tumour was encapsulated and stripped off the kidney with ease, leaving it normal, with no suggestion of its having any association with the mass. After operation the blood pressure fell to 120/80, and the patient seemed weak and collapsed. He developed a basal bronchopneumonia and died on June 13, six days after operation. Post-mortem examination was not allowed.

Case 2

A woman aged 57, under the care of Mr. G. T. Mullally, was first seen at Westminster Hospital on August 2, 1946, complaining of a painless swelling in the right upper abdomen which had been present for 3½ years. She had no symptoms apart from occasional abdominal colicky pains and a mild degree of constipation.

Examination showed that she was well nourished and of slight build, with a blood pressure of 130/80, and no clinical suggestion of any hormonal abnormality or of Cushing's syndrome. A hard rounded mass was palpable in the right upper quadrant of the abdomen. It was not attached to the skin, and moved down on respiration. A barium meal revealed displacement but no abnormality of the bowel. Excretion pyelograms showed normal renal shadows, and the blood urea was 34 mg. per 100 ml.

Laparotomy, performed by Mr. Mullally on August 18, revealed a large encapsulated retroperitoneal tumour lying slightly behind and to the right of the right kidney, but in no way associated with it. No adrenal gland could be found on the right side. The tumour was easily removable, but after operation the patient was collapsed, with depressed blood pressure of 80/40. Plasma and blood transfusion produced no improvement. On the fourth day a basal bronchopneumonia developed, and the patient died on the sixth day after operation. Post-mortem examination was not allowed.

Morbid Anatomy and Histology

The morbid anatomical and histological appearances of the tumours were so similar that they may be described together.

In each case roughly spherical completely encapsulated tumours were found, with the following dimensions and weight :

	Diameter	Weight
Case 1	22.5 cm. (8¾ in.)	3,750 g. (8¼ lb.) (see Fig. 1)
Case 2	23.5 cm. (9¼ in.)	5,350 g. (11¾ lb.) (see Fig. 2)

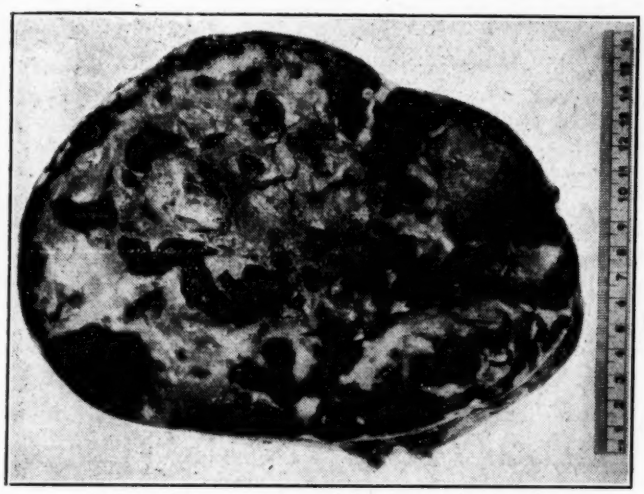

FIG. 1.—The cut surface of the tumour in Case 1.

There was no evidence of a pedicle or of any association with the kidney, and in neither case was an adrenal gland found on the same side as the tumour. The capsules were quite thin but extremely tough. The cut surface of the tumour showed considerable haemorrhage and necrosis, so that most of the substance consisted of pinky-yellow friable material, leaving only small areas of yellowish fleshy tumour substance lying just beneath the capsule. Spaces containing blood clot were a noticeable feature of both cases.

Microscopically the central part of the tumours was found to be composed mostly of necrotic debris with occasional groups of cells remaining. Only in those areas lying immediately under the capsule was it possible to determine the histological pattern. In each case the picture was similar, and consisted of groups of polyhedral or round cells with deeply

FIG. 2.—The cut surface of the tumour in Case 2.

staining clear-cut nuclei and foamy pale-staining cytoplasm, bounded by a well-marked cell membrane. These cells were arranged principally in columns, but often enclosed spaces. The general appearances were similar to those found in normal adrenal cortex (see Fig. 3).

Frozen sections of the tissue stained by methods to demonstrate fat showed the cytoplasm of the cells to be packed with lipoid globules. Fuchsinophil staining by Broster and Vines's (1933) method gave for the most part negative results, although occasional granules scattered through the cells of the tumours took up the red stain feebly.

A marked feature of the tumours was their extreme vascularity, and large numbers of small vessels had cells of cortical

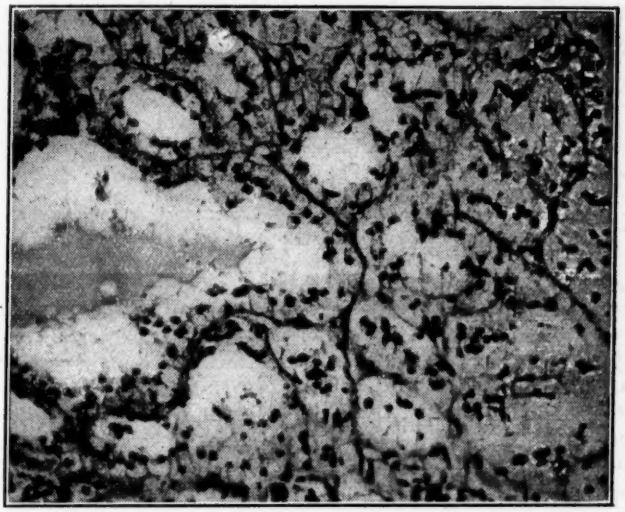

FIG. 3.—Typical microscopical picture at periphery of tumour (Case 1). (× 65.)

suprarenal type either closely attached to or actually forming their walls (see Fig. 4). Many areas of both tumours showed evidence of thrombosis in vessels.

In the necrotic areas there were small granular zones of calcification, not large enough to cause noticeable "grittiness" on cutting, but demonstrable microscopically by special staining methods. This feature is considered to be evidence that the

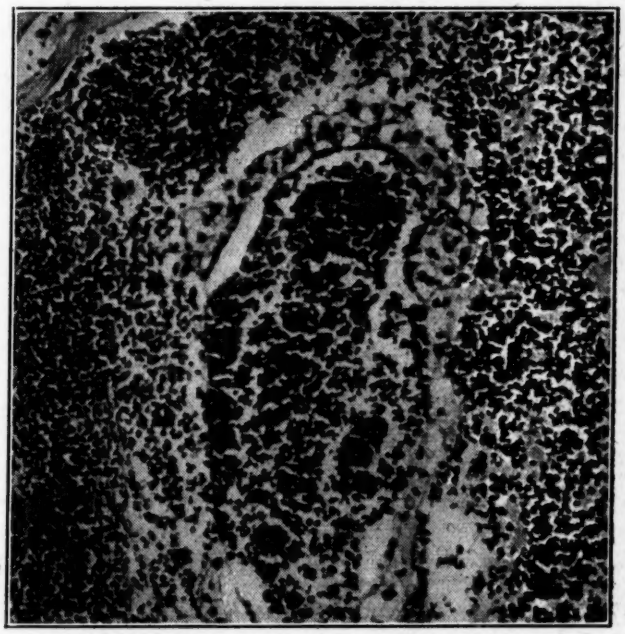

FIG. 4.—Photomicrograph showing close association of tumour cells with a small vessel (Case 2.) (× 65.)

breakdown of the tumour was not a very recent occurrence. The capsule was thin and in each case made up of fibrous tissue with intermingled smooth-muscle fibres.

Careful search of both tumours failed to reveal any of the cytological features usually associated with malignancy, such as giant-cell forms or hyperchromatism of the nuclei.

Discussion

These cases present interesting features both clinically and pathologically. Laparotomy was essential in order to make a diagnosis, and easily removable tumours were found, but in both cases blood pressure fell sharply afterwards, suggestive of suprarenal shock, and it seems probable that this feature was in part, if not entirely, the cause of death. It is therefore important to consider the possibility of a cortical suprarenal neoplasm when a very large encapsulated retroperitoneal tumour is found which is causing no symptoms referable to the organ of origin; for it seems likely that if the diagnosis were made early enough hormonal replacement therapy might prevent post-operative collapse. It is therefore particularly valuable to note that the naked-eye appearances in my two cases and in the case described by Anderson *et al.* (1943) were exactly similar.

Walters and Kepler (1938) pointed out that probably the most important single feature in the surgical treatment of cortical adrenal tumours was the control of post-operative shock. Cecil (1933) stated that 39% of patients operated on died of shock. It may be added in this connexion that Cahill *et al.* considered that post-operative shock occurred only in those cases in which, as a result of the tumour, a syndrome of Cushing's type was present and hormonal excess was affecting metabolic functions such as salt-and-water balance and capillary permeability. They further state that, in their series, cases without clinical hormonal upsets were never associated with post-operative collapse. This view cannot be confirmed from the present cases.

From the pathological point of view the gross and the microscopical structure of these tumours provokes discussion whether they are benign or malignant. Features in favour of their benignity are the presence of the well-formed capsule and the absence of evidence of metastases, although it must be admitted that it was not possible to perform necropsies. The absence of demonstrable mitoses and the close resemblance of the tumour cells to those of the normal adrenal seem to point to a non-malignant origin, although it may be repeated that numbers of metastasizing tumours with complete differentiation of cell type have been described.

It is somehow difficult to reconcile such very large masses showing so much necrosis and haemorrhage with benign growths. Another feature suggestive of malignancy is the association of tumour cells with the vessel walls, and in many cases the presence of strands of cells lying free in blood spaces. The marked tendency towards thrombosis in the vessels is more commonly seen in malignant than in benign tumours.

Examples in the literature of large cortical adrenal tumours are rare. Among the largest is the case recorded by Holmes (1925) in which a tumour weighing 1,025 g. (2 lb. 4 oz.) and measuring 17 by 14 by 9.5 cm. was found to be of benign type, and that of Anderson *et al.* (1943), who described a malignant cortical tumour weighing 1,780 g. (3 lb. 14 oz.) in which a solitary metastasis was found in the liver. The latter case is of interest, as the appearance of the tumour was identical with those described above, although a relatively anaplastic histological pattern was recorded.

It may again be stressed that histologically benign tumours merge imperceptibly into the malignant varieties, and in the absence of demonstrable metastases opinions might well differ about their correct interpretation. It might be argued that haemorrhage into a simple adenoma is the more correct view in the above cases, but it is my opinion, based on the reasons already given, that these are examples of slowly advancing malignant tumours.

Summary

Two cases of very large cortical adrenal tumours are presented.

The complete absence of localizing clinical signs is noted.

The importance of their recognition in order to institute post-operative treatment is stressed.

The question of their benign or malignant origin is discussed.

My grateful thanks are due to Mr. G. T. Mullally and Sir Stanford Cade for permission to make use of the case records.

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A SECOND EXAMPLE OF THE "DUFFY" ANTIBODY

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AND

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(Blood Group Reference Laboratory, Ministry of Health and North London Blood Supply Depot, National Blood Transfusion Service)

Cutbush, Mollison, and Parkin (1950) have described an antibody which defines a new blood-group system. The antigen (Fy^a) distinguished by this antibody was found to be present in 66% of the English population, and appeared to be independent of the *ABO* and *Rh* groups of the blood tested. The antibody, anti-Fy^a, was found after a transfusion reaction in a haemophilic patient who had received previous transfusions. It was accompanied by the *Rh* antibody anti-*D*, which could, however, be removed by absorption. The reactions of the new antibody could be detected with certainty only by means of the Coombs test.

A second example of the same antibody has been detected in the serum of Mr. J. S. In or about the year 1943 he had twice been transfused for haemorrhage from a peptic ulcer. In August, 1949, he was again admitted to hospital for the same condition. His blood was found to be group *AB*, and two bottles of group *B* blood were directly matched against his serum at the hospital. He had a severe rigor after receiving about 20 ml. of blood from one bottle. The transfusion was interrupted and begun again from the other bottle with a similar result. It unfortunately proved impossible to trace the donors of these bottles of blood.

A full investigation of the patient's blood was then carried out. He was found to be group A_2B, R_1R_1 (*CDe/CDe*), and his serum, though containing no saline agglutinins, was found by means of the Coombs test to contain an antibody which reacted with more than half of the red-cell samples tested. It was almost immediately

found to be giving similar reactions to the newly discovered anti-*Fy*^{a}. A detailed comparison has shown that of 62 *Duffy*-positive bloods all were positive with the serum of J. S. Of 29 *Duffy*-negative bloods all were negative or reacted very weakly with the serum of J. S. As the *Duffy* reactions of many of these bloods were previously known the numbers given do not constitute evidence of the frequency of the antigen in the general population. In the course of these tests the serum of J. S. was found to contain a trace of anti-A_1, which was easily removed by absorption with A_1B, *CDe/CDe Duffy*-negative cells. A further trace of one or more antibodies was also detected, consisting probably of anti-*E* and possibly of anti-*c* as well. Only in three cases did these antibodies give a reaction as strong as that of the anti-*Fy*^{a}. As we had used up all our serum and no more could then be obtained from the patient it was impossible to investigate these antibodies further. It is, however, clear that the main antibody in his serum is anti-*Fy*^{a}, and it is probable that this was the cause of his transfusion reactions.

We wish to thank Dr. H. Rogers of Hillingdon Hospital, who referred this case to us and gave us much assistance; and Dr. R. R. Race, Dr. P. L. Mollison, and Miss Marie Cutbush, who carried out many of the comparative tests of the two sera.

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Medical Memoranda

Recovery from Strangulated Obturator Hernia

Obturator hernia commonly presents in elderly women and is usually diagnosed as intestinal obstruction or strangulation. The patient is emaciated and dehydrated—a poor subject for surgery. Therefore it is hardly surprising that the mortality is high. Watson (1939) collected 149 cases operated on, with a fatality rate of 48%. Meyer (1914) recorded a fatality rate of 54% and Grasser (quoted by McMahon, 1915) one of 81%, while Horine (1927) reported 47 cases with 29 recoveries, a fatality rate of 38%.

In view of the comparative rarity of the condition the following case is recorded.

CASE REPORT

On May 15, 1947, a woman aged 70 was admitted to hospital complaining of abdominal pain and vomiting of seven days' duration. Vomiting had been projectile in character and, latterly, faecal. She had passed no flatus for several days.

On examination she was seen to be a very thin old woman, somewhat disorientated, and a poor witness. Her tongue was dry and furred, and she was vomiting faecal-looking material. The abdomen was distended, especially centrally, and there was gross visible peristalsis. Tenderness in the right iliac fossa and rebound tenderness were present. Rectal examination was negative. A fair range of painless movement was possible at both hip-joints. A diagnosis of low small-bowel obstruction with, possibly, internal strangulation was made.

After four hours of continuous suction and intravenous saline her general condition improved slightly, though the physical signs in the abdomen remained unchanged.

Operation was performed under local analgesia with minimal thiopentone and cyclopropane. Greatly distended coils of small bowel were found, one of which was trapped in the right obturator foramen. The opening in the obturator membrane was stretched and the loop was withdrawn; though of a dark-purple hue, it was viable. The sac was evaginated and removed, and the opening in the membrane was closed with one silk mattress stitch.

Convalescence was uneventful, except that the patient was extremely confused mentally for several days. She was discharged well eleven days after operation.

In view of the length of the history—seven days—and the age of the patient it is remarkable that she did so well. At no time did she complain of pain radiating down the inner side of the thigh to the knee (Romberg-Howship sign), but her condition on admission was so bad that she was unable to give a good account of herself.

My thanks are due to Mr. James Troup, senior surgeon, Royal Hants Hospital, Winchester, for permission to publish this case.

M. C. WATERFALL, F.R.C.S.,
Formerly Resident Surgical Officer, Royal Hants Hospital, Winchester

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Tuberculous Meningitis during Pregnancy, Streptomycin Treated

During the past two years in a series of 84 cases of tuberculous meningitis receiving streptomycin treatment at this hospital two patients developed their symptoms during pregnancy.

CASE 1

This patient, aged 21 years, had been ill for ten days with acute meningeal symptoms when admitted to this hospital on November 9, 1948. She was then at the 29th week of her first pregnancy and weighed 167 lb. (75.75 kg.). Shortly before her marriage she had been detected at her works radiographic survey as a possible early pulmonary case, but had neglected her follow-up arrangements. The pregnancy had been uneventful until the onset of her meningeal symptoms. The uterus corresponded to her period of amenorrhoea and the foetal heart was heard. She presented the ordinary clinical and cerebrospinal fluid findings of a tuberculous meningitis, and in spite of an active chest condition she was given streptomycin treatment at once. The diagnosis was subsequently confirmed by the culture of a human strain of tubercle bacillus from her fluid. She was given 100 mg. of streptomycin intrathecally daily for ten days, and then on alternate days for ten doses. Intramuscularly she received 1.6 g. daily, given in two equal doses at intervals of twelve hours. This was at the rate of 10 mg. per lb. (22 mg. per kg.) of body weight per day.

This treatment produced a good response and the meningeal condition improved, while there seemed to be no interference with the pregnancy. Serum streptomycin content varied from 2 to 4 μg. per ml. and cerebrospinal fluid streptomycin was from 2 to 4 μg. per ml.

On January 18, 1949, in her 39th week of pregnancy, she came quickly into labour and delivered herself spontaneously of an apparently normal female infant of 6 lb. 15½ oz. (3.16 kg.). She was transferred to the Royal Maternity Hospital, Belfast, and returned to us a week later. There was no break in her intramuscular medication. At the time of her delivery, ten weeks after starting streptomycin treatment, she had received 2 g. intrathecally and 112 g. intramuscularly. I have not seen the child, but I understand from Professor F. M. B. Allen, of the Department of Child Health, that it is apparently normal and healthy. He found it to be Mantoux-negative, and arranged for it to have B.C.G. vaccination before release of the mother from hospital.

The patient was given a further course of intrathecal treatment of 30 doses of streptomycin after her delivery. The cerebrospinal fluid of January 31 gave a culture of tubercle bacilli. Improvement has continued: intramuscular streptomycin was stopped on July 13 and she remains very well. She has recently been sterilized, with a view to returning home on completion of her infant's B.C.G. vaccination.

CASE 2

This patient, aged 29, was at the 15th week of her third pregnancy when admitted here on March 31, 1949, having been ill with meningitis for ten days. Her cerebrospinal fluid was clear and showed protein 120 mg.%; cells 147 (all lymphocytes); sugar 30 mg.%; chlorides 703. These findings were accepted as diagnostic of tuberculous meningitis, but the organism was not recognized or recovered on culture. Her pregnancy had apparently been normal, and she weighed 123 lb. (55.79 kg.). Treatment with streptomycin was at the rate of 100 mg. intrathecally daily and 600 mg. intramuscularly 12-hourly. Her meningeal condition responded well and she continued to feel foetal movements, although foetal heart sounds were not heard.

On April 11 she had a blood streptomycin level of 6 μg. and a cerebrospinal fluid content of 8 μg. At this stage Mr. H. I. McClure examined her, and considered her pregnancy a normal one of about 16 weeks' duration.

On April 22 her uterus emptied spontaneously, and she admitted that she had missed foetal movements for the previous two days. She was severely shocked, and curettage was carried out by Mr. McClure. Her recovery was uneventful, and she was discharged from hospital on August 4, apparently completely recovered.

Dr. J. E. Morison, of the Institute of Pathology, reported upon the uterine contents as follows. "Foetus and membranes correspond to about 18 weeks' pregnancy. It is possible to exclude by gross examination any congenital malformation of the heart or great vessels. The lungs and brain seem to be developing normally. There is most extensive calcification of the chorionic villi. At this stage the appearances are very unusual: it is not possible to regard them as distinctive or specific and similar, though less extensive changes have been seen in other abortions where no pathology could be demonstrated. The relationship of the changes to the therapy with streptomycin must await further cases or reports from other centres. On the whole, any specific connexion appears unlikely."

Apart from the colleagues mentioned, I am also indebted to Dr. G. F. W. Tinsdale for laboratory records and to Dr. L. V. Reilly for the streptomycin levels and typing of the organisms. Dr. F. G. Grant, of the Royal Maternity Hospital, Belfast, is at present preparing a thesis on this material for his M.R.C.O.G.

F. F. KANE, M.D., M.R.C.P.I., D.P.H.,
Medical Superintendent, Northern Ireland Fever Hospital, Belfast.

Examination of Young Children with Acute Appendicitis

Acute appendicitis is not a common illness in children under the age of 4. When this diagnosis is suspected in a very young child the examining physician or surgeon is faced with two difficulties. First, the child is not able to give a concise history or accurately to locate its pain. The parent usually states that the child has suddenly become irritable and peevish, refused its food, and perhaps vomited once. There is generally some alteration in bowel habit either in the form of constipation or the occurrence of one rather loose and copious motion followed by constipation. The manifestation of pain is limited in the early stages to irritable crying and fretfulness, with the occasional passage of the child's hand across its abdomen. Fortuitous pressure on the abdomen while the child is being comforted causes more pronounced crying and fretfulness, without the necessary explanation.

The second difficulty is in the adequate physical examination of the abdomen. With the child in the recumbent position and able to see what is intended, any approach to the abdomen causes such resentment and apprehensive crying that palpation in order to assess localized tenderness or rigidity becomes almost impossible. To wait by the bedside for a more favourable moment does not usually help matters, for the pain of acute appendicitis is constant, and each fresh approach to the abdomen is resisted by the child. This is in sharp contrast with the events in cases of intussusception, in which, the pain being intermittent, it is usually possible by awaiting a favourable moment to carry out a satisfactory palpation of the abdomen in between attacks of colic.

I have been so baffled by this difficulty in palpating the abdomen in several children under the age of 4 who were in fact suffering from acute appendicitis that I have evolved the following method of physical examination in such cases, with results which have so far rendered positive diagnosis possible. I claim no originality for this method, but have found it so useful on occasions that I place it on record in the hope that others who may not have tumbled to it may find it helpful.

METHOD

The child is held by the mother or nurse in the position in which a fretful child is comforted normally—that is, clasped to its mother's breast with its arms and legs embracing its mother.

Photograph showing position in which the child is held.

From behind, the examiner then places the palms of both hands on the anterior-superior iliac spines, with the fingers extended across the child's abdomen in the direction of the umbilicus (see photograph), but no pressure is exerted by these fingers in the first instance. The child usually gives a wriggle of resentment when the hands are first applied, but as no pain is caused and as it cannot see what is happening this resentment soon passes off. The hands are maintained in position until the child is quiet, and then gentle but increasingly firm pressure is exerted by the extended fingers overlying the *left* iliac fossa. This again causes resentment and irritability, which subsides in a short while. Similar pressure is then exerted in the *right* iliac fossa by the fingers of the other hand while easing the pressure on the left. In positive cases the contrast is striking. Instead of a wriggle of resentment the child gives a sharp cry of pain, and, if the pressure has been applied gradually, local muscular rigidity can be felt before it is masked by the generalized rigidity which results from crying.

It is not always possible to carry out this examination more than once, as the child becomes wise to events. But, carefully done once, the signs are so positive that operation can be confidently recommended on the findings.

ROBERT STRANG, Ch.M., F.R.C.S

Reviews

TOWARDS DIAGNOSTIC PRECISION

Clinical Diagnosis by Laboratory Examinations. By John A. Kolmer, M.S., M.D., F.A.C.P. Second edition. (Pp. 1,212; illustrated. $12.) New York: Appleton-Century-Crofts, Inc. 1949.

An Atlas of the Blood and Bone Marrow. By R. Philip Custer, M.D. (Pp. 321; 285 illustrations, 42 in colour. £3 15s.) Philadelphia and London: W. B. Saunders. 1949.

A visitor to the United States recently heard one resident (*anglice*, registrar) ask another, "How do you diagnose disseminated sclerosis?" The correct answer was not, as might have been expected, Charcot's triad, but a zone I reaction in the colloidal gold test. This kind of thing makes English clinicians pharisaically delighted that they are not as the Americans, but, having observed the tendency to fashions in clinical diagnosis in this country, I am not sure that the fault is all on one side.

Dr. Kolmer has been both a professor of pathology and a professor of medicine, and is therefore well fitted to put things in a proper perspective:

> "No one can have a higher respect and reverence than I for the great physicians of the past who, perforce, relied alone on the art of medicine. But if they could walk our wards to-day they would be among the first to confess their sins of commission in diagnosis while voicing a prayer that all of our present and future knowledge in both the art and science of medicine will continue to reduce progressively the errors of diagnosis revealed in the postmortem room and due, not so much to errors of commission, as to those of omission so frequently ascribable to lack of knowledge, carelessness, or indifference."

His book is essentially a textbook of medicine written from the laboratory viewpoint. I have tried it out with one or two recent final examination papers in medicine in this country and have been surprised to find how large a proportion of the questions could have been answered from a study of this book alone. Nearly 1,000 pages are allotted to medical text and only 150 to the technique of laboratory examinations. The index comprises, at a rough computation, about 12,000 items, and there are numerous references, dating up to 1948, at the end of each chapter. This is indeed an "immense and glorious work of fine intelligence." It is smooth reading and gives not only an account of the pathological changes in disease which are relevant to diagnosis by laboratory methods but also practical details, often illustrated by photographs.

It is the kind of book I should like to possess, but I am not so sure about my students. The publishers say that it is essentially a practical guide to the diagnosis and differential diagnosis of disease through the interpretation and practical application of the various laboratory tests and methods, but is the student really to study the whole of Price's *Medicine*, the whole of Kolmer, and at the end of it all still be largely ignorant of psychology, psychiatry, social medicine, and general practice? Even the Americans, with their assignment system of teaching, are finding the load too great and are replacing formal textbooks in clinical pathology by practical manuals of multiple authorship, such as the *Syllabus of Laboratory Examinations in Clinical Diagnosis*, which the Harvard University Press is now publishing. It may well be, then, that Kolmer is a period piece, but it is certainly a magnificent one and well worthy of acquisition.

An Atlas of the Blood and Bone Marrow is the most welcome book on haematology which has appeared since the first edition of Wintrobe's *Clinical Haematology*. Dr. Custer accumulated much of his material during eighteen years as a hospital pathologist specially interested in haematology. In the recent war he was in charge of the division of reticulo-endothelial pathology of the American Army Institute, which received an unbelievable amount of material. Since the war he has taken part in the discussions on haematological nomenclature which have been held under the aegis of the American Society of Clinical Pathologists and the American Medical Association.

Dr. Custer is therefore neither an amateur nor a peripheral haematologist, and the book represents the wide range of his knowledge. It gives a short account of the diseases of the blood, but the main emphasis is on the illustrations. Notable points are the full description of the anatomy and embryology of blood formation, the emphasis on bone-marrow sections, and the use of bas-relief for demonstrating the shape of the red cells. The text is on normal lines, brief and dogmatic, omitting all descriptions of technique except in so far as examination of the marrow is concerned. Indeed, for an American book it is surprisingly undocumented, being based almost entirely on personal experience. Dr. Custer has the gift of saying a great deal in a few words, and the sections on myelophthisis and on malignant lymphoma, though they represent his personal views, are masterpieces of compression.

The illustrations, of which there are nearly three hundred, are magnificent. Nearly all are photographs, most in black and white, some in colour. The book is printed on a large page, and the full-page pictures are approximately 6 by 8½ inches (15 by 21 cm.) I have never seen anything to equal the quality of these illustrations. The black-and-white plates are superb, sharply focused and definitive. The coloured photographs, though a little less crisp in focus, are magnificent achievements. I must ask any reader who thinks my recent criticism of Israël's *Atlas* was too scathing to compare these two books. Messrs. W. B. Saunders are greatly to be congratulated on the beauty and speed of production. This is indeed a book to have and to hold.

L. J. WITTS.

PHYSIOLOGY AND ANAESTHESIA

Resuscitation and Anesthesia for Wounded Men. The Management of Traumatic Shock. By Henry K. Beecher, A.M., M.D. (Pp. 161; illustrated. £2.) Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications.

Fundamental Considerations in Anesthesia. By Charles L. Burstein, M.D. (Pp. 153; 64 figures. £1 10s.) London and New York: The Macmillan Co. 1949.

Dr. Beecher's book is largely the outcome of the author's experiences when he was consultant in resuscitation and anaesthesia to the U.S. Army in the Mediterranean zone during the war. Many of the sections have been left as they were written at the battlefront, and much of their pungent vividness comes from this fact. The theme is the treatment of soldiers from the moment of wounding up to the time of their operation, and continues with discussion of the best type of anaesthesia to employ.

The first section is concerned with the mental and physical state of the patient. The functions of the Army chaplain when attending a mortally wounded soldier are discussed somewhat vaguely. Section two describes the variations from normal physiology found in wounded men by

biochemical tests. Section three gives a good account of resuscitation by modern methods, and the book closes with a description of the most suitable anaesthetic techniques for field surgery.

Although war wounds only are considered, the work can be warmly recommended to casualty officers or house surgeons, who are often called upon to treat patients suffering from road accidents or industrial injuries. Furthermore, British anaesthetists are not likely to question the author's remark in the preface that "if war should come again, the civilian as well as the soldier will be in the thick of it." The book is profusely illustrated with excellent photographs, mostly taken in the battle area. There is also one rather macabre coloured plate.

Fundamental Considerations in Anesthesia is a notable addition to the physiology and pharmacology of anaesthesia which contains the observations of a practical anaesthetist and the results of much original work. Dr. Burstein sets out his views simply and clearly, though they do not always coincide with accepted teaching. He attaches great importance to the carotid sinus reflex, and allots a whole chapter to it: British anaesthetists have tended (perhaps wrongly) to regard this reflex as mainly of theoretical interest. Controlled respiration is considered at some length, but it is strange to read of seven conditions useful in producing it, carbon dioxide depletion being relegated to the last. It is doubtful if the Madison school would agree with this.

The chapter on shock is excellent, but universal acceptance would not be forthcoming in Great Britain of the statement that "the use of pentothal sodium or other intravenous barbiturates is now condemned in the treatment of the shocked patient." Intravenous procaine is referred to many times in particular doses, but the dangers of rapid injection are not mentioned nor the fact that an immense total dose can be given provided the blood concentration is kept low by limiting the injection rate to under 20 mg. per minute.

These random observations are not intended as criticisms, and indeed differences of opinion are valuable in that they stimulate the reader to think out problems for himself instead of accepting statements at their face value. The text has not been anglicized, and contains such expressions as "explication," "reactivity," "compensated respiration," and "mid-duction of the vocal cords." The book is well produced, with clear illustrations, but it must be admitted that the price is high for under 150 pages of reading matter. This is presumably due to the present asthenia of sterling.

C. LANGTON HEWER.

FIELD EPIDEMIOLOGY

Epidemiology in Country Practice. By William Norman Pickles, M.D. With preface by the late Professor Major Greenwood, F.R.S., D.Sc., F.R.C.P. Reissued 1949. (Pp. 112; illustrated. 10s. 6d.) Bristol: John Wright and Sons.

Dr. Pickles's notable book on field epidemiology was first published in May, 1939, but the entire bound and sheet stock and the type were destroyed by enemy action in 1941. To meet the continuing demand it has been reset and reissued. In the interval Dr. Pickles has lectured widely in this and other countries, and many will welcome the opportunity to read the story which they have heard him tell in his own inimitable fashion.

The object lesson of this work is that a general practitioner in the course of his practice over a long term of years can make observations of such accuracy that their sum is a substantial contribution to scientific knowledge. This may sound platitudinous, but it is true that, with notable exceptions in epidemiology—as in the work of William Jenner, John Snow, and William Budd—the opportunities have been largely missed. Yet there are many practitioners who question the pronouncements of their teachers and statements passed on from textbook to textbook and still fail to record systematically the facts which have raised their questionings. This systematism and precision are the keynotes of Pickles's work. Something happens which sets him thinking and questioning. He remembers an account in a medical journal or some old monograph. He forms a tentative opinion and waits for the probable recurrence of the event which will challenge or confirm his earlier conclusions. At each stage the facts are carefully noted and, if suitable, treated by simple statistical methods such as charting in order to clarify their presentation.

To those who have attacked epidemiological problems with a combination of hospital observations and the handling of mass data—much of them based on observations of very varying reliability—Pickles's careful studies are a comfort and an illumination. He would be the first to admit that conclusions drawn from relatively small numbers of observations carry their own risks. For instance, his contention that incubation periods vary less than we have supposed might have to be modified if it were possible for one man to observe a very large number of instances of short exposure. It is only with large numbers that the greater deviations from the usual are to be expected. On the other hand, few of the short or long incubation periods recorded by others are based on as careful examination as those of Pickles.

Space does not permit more than a passing reference to Pickles's pioneer work on epidemic catarrhal jaundice and epidemic myalgia. The vivid account of sufferers from the latter disease is an example of the delightful style in which this book is written. It should be in the library of every practitioner of medicine, and to young men entering rural practice it is indispensable.

RALPH M. F. PICKEN.

THE LIFE TABLE

Length of Life. A Study of the Life Table. By Louis I. Dublin, Ph.D., Alfred J. Lotka, D.Sc., and Mortimer Spiegelman, F.S.A. Revised edition. (Pp. 379. $7.00.) New York: The Ronald Press Company. 1949.

The life table based on current mortality, and its best-known function the expectation of life, is a fiction useful in demonstrating certain mortality trends but particularly unreal where mortality rates are declining and the population is not stable in its age-distribution. Standardized death rates, or better still age and sex specific rates, are as valuable a criterion of the public health and far easier to calculate, and it is unfortunate that the authors give the expectation of life so much prominence. The increases in the expectation of life in this century are better portrayed as major decreases in mortality in the early years of life. Mortality of old people and the life span have changed little in the same period, so that the major social and medical problem of an ageing population is one of an increasing proportion of older people and not one of an increase in longevity.

The description of the life table is excellently written, and the chapters on the factors influencing mortality and longevity have that rare gift of being informative to the professional and interesting and understandable to the layman. Most of the data refer to the U.S.A., but the

principles and trends illustrated apply generally to English vital statistics and would serve as an introduction to more detailed publications.

This book first appeared 14 years ago, and in this revision the material has been brought up to date and two new chapters have been added—" Biological Factors Influencing Longevity and Mortality " and " Forecasts of Mortality and Longevity." This latter introduces generation life tables and follow-up studies which may well be of increasing importance with the new interest in chronic disease. This volume maintains throughout the high standard associated with publications of members of the statistical bureau of the Metropolitan Life Insurance Company.

J. KNOWELDEN.

FUNDAMENTALS OF CHEMISTRY

Topics in Physical Chemistry. By W. Mansfield Clark, Ph.D., Sc.D. (Pp. 738; 300 figs. £2 4s.) London: Baillière, Tindall and Cox.

The particular aim of this book is the education of understanding as distinct from the plain and simple knowledge of facts. The author, in his experience as professor of physiological chemistry, has been impressed by the frequency of gaps in knowledge of one or other of the fundamentals of science manifested by students entering on the curriculum and that among entrants whose earlier training was looked upon as complete and exhaustive. Such gaps are a barrier to the kind of education in physical chemistry that he desires to inculcate, and the book is a good foundation rather than a text for a formal course.

The author recognizes a danger which arises from the effort to keep pace with the rapid advancement of special sections of science, that the pursuit of special sections results in a neglect of some of the basic principles which are essential to the complete background. He sees two sides of the problem: one is the facility of acquiring knowledge from the direct enunciation of principles with instances of practical application; the other is the idea expressed by Whitehead in the dictum that " the utmost abstractions are the true weapons with which to control our thought of concrete fact." He recognizes that these two trends follow divergent paths, but has nevertheless succeeded in keeping both in perspective. As a result the treatment of his topics is well balanced, the description of phenomena clear, and the discussion of inter-relationships illuminating. The work will prove a valuable adjunct to the textbooks in use by students and a most useful book of reference for those whose interest is continued after graduation.

T. TICKLE.

In *Biochemistry in Relation to Medicine* (pp. 442; illustrated; £1 5s.; London: Longmans, Green and Co., 1949) Dr. Carter and Professor Thompson aim " to present all those aspects of biochemistry which are significant in medicine." They begin with an introductory account of physical chemistry and a brief but adequate description of the chemistry of carbohydrates, lipids, and proteins. There follows an outstandingly good section on enzymes and biological oxidations, on which the writers are authorities. In the chapters on haemoglobin, electrolytes of the body, acid-base equilibrium, and carbohydrate metabolism both physiological and pathological aspects are considered. There is an interesting discussion on the chemistry of food, nutrition, and vitamins: the remaining chapters describe the chemistry of bile, urine, and plasma proteins and the chemical functions of the stomach, liver, pancreas, and kidney. The outstanding feature of the work is the authors' clear insight into the medical student's needs and limitations. It is a book of biochemistry in its true sense, for it gives an account of the chemical reactions of living cells rather than a description of the organic chemistry of dead tissues, which often passes for medical biochemistry. It is written in an interesting, easily readable style, there are many references to the literature, and a valuable section on practical work is given at the end of almost every chapter.

BOOKS RECEIVED

Review is not precluded by notice here of books recently received

An Autobiography. By Sir A. Keith. (Pp. 721. 25s.) London: Watts. 1950.

Gardening in East Africa. Edited by A. J. Jex-Blake, M.D., F.R.C.P. 3rd ed. (Pp. 399. 30s.) London: Longmans, Green. 1950.

Industrial Nutrition. By M. Pyke, B.Sc., F.R.I.C. (Pp. 205. 10s.) London: Macdonald and Evans. 1950.

The Diagnosis of Hysteria. By D. W. Abse, M.D., D.P.M. Foreword by J. H. Ewen, F.R.C.P.Ed., D.P.M. (Pp. 112. 8s. 6d.) Bristol: John Wright. 1950.

Operative Surgery. Edited by A. Miles, M.D., F.R.C.S.Ed., and Sir James Learmonth, K.C.V.O., C.B.E., F.R.C.S.Ed. 3rd ed. (Pp. 559. 30s.) London: Geoffrey Cumberlege. 1950.

Very Peculiar People. By E. J. Dingwall, M.A., D.Sc., Ph.D. (Pp. 224. 18s.) London: Rider. 1949.

Advances in Surgery. By W. DeWitt Andrus and others. (Pp. 590. 88s.) Vol. 2. London: Interscience Publishers, Ltd. 1949.

Illness in Infancy. By R. M. Dykes, M.D., D.P.H. Foreword by F. Grundy, M.D. (Pp. 46. 3s. 8d.) Luton: The Leagrave Press. 1950.

The University of Queensland Papers. Personality Deviations Occurring in Children of Pre-School Age. By J. Bostock and E. Hill. (Pp. 76. 7s. 6d.) Brisbane: The University of Queensland Press. 1949.

The Practice of Urology. By H. C. Rolnick, M.D. Vols. 1 and 2. (Pp. 1,245. 189s.) Philadelphia, London, and Montreal: Lippincott. 1949.

Antibiotics. By R. Pratt, Ph.D., and J. Dufrenoy, D.Sci. (Pp. 255. 40s.) London, Philadelphia, and Montreal: Lippincott. 1949.

Skin Grafting. By J. B. Brown, M.D., and F. McDowell, M.D. 2nd ed. (Pp. 339. 60s.) London, Philadelphia, and Montreal: Lippincott. 1949.

Surgery of Repairs. By J. F. Pick, S.B., M.D., F.I.C.S. Vols. 1 and 2. (Pp. 828. 189s.) London, Philadelphia, and Montreal: Lippincott. 1949.

Brucellosis (Undulant Fever). By H. J. Harris, M.D., F.A.C.P., and B. L. Stevenson. Foreword by W. M. Simpson, M.S., M.D. 2nd ed. (Pp. 617. $10.00.) New York: Paul B. Hoeber. 1950.

A Pattern for Hospital Care. By E. Ginzberg. (Pp. 368. 36s.) New York: Columbia University Press. London: Geoffrey Cumberlege. 1949.

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MEDICAL ASPECTS OF CRIME

The increase in crime since the end of the war has been even more than was expected. Grave public concern has been aroused, especially by the fact that so many of the offenders have been boys and adolescents. At first sight this would appear to be an omen of danger: if the youth of the country is turning to evil ways the future of society is imperilled. A recourse to statistics, however, supplies a useful corrective. In his invaluable and comprehensive survey, *Society and the Criminal*, Sir Norwood East publishes an analysis by age of the offender of the criminal statistics for England and Wales for the years 1929–38.[1] Expressed as a proportion of that part of the total population at the same age, the number of those who commit crimes reaches its peak at 13 per 1,000 among boys of 13, but after that drops rapidly. It is halved by the age of 20, and halved again after 30. It was therefore only to be expected that the post-war outbreak of criminality should show itself most plainly in the most susceptible age group. The fact that juvenile delinquency is so common is by itself insufficient evidence that the times are corrupt or that parents are seriously neglecting their duties. The rises and falls in the incidence of crime which occur with time and with social changes are only one among the many reasons for thinking that delinquency is much more a social than it is a medical phenomenon. The medical profession would lose its reputation for balanced judgment if it were to claim that doctors and psychiatrists alone have all the answers to this problem, or even the most important ones. Caution is especially required when prevention and treatment are discussed. The atmosphere of this debate is charged with emotion: on the one side there are the advocates of severer punishment and the reintroduction of the "cat," and on the other those who maintain that all that is needed are more and more child-guidance clinics. Half-way between, but still lacking the support of conclusive evidence, are those who believe that criminality is encouraged by a superfluity of laws and regulations without a moral sanction, and that it is certainty of detection rather than severity of punishment which acts as a deterrent.

[1] *Society and the Criminal*, 1949, London: H.M.S.O.
[2] *Bull. World Hlth Org.*, 1949, **2**, 279.
[3] *Law and Contemporary Problems*, 1935, **2**, 436.

The point made by Dr. John Bowlby in the article appearing in this issue, that before social measures and other remedies are offered they should be based on properly controlled research, is therefore a cogent one. There is no reason why the research should be exclusively medical, but plainly medical investigations have their part to play. Even the raw statistics point to promising, but neglected, lines of inquiry. Why is it that crime is so much commoner in the male than in the female? And why is it that the proportion of males to females sinks steadily with age, instead of varying with sexual maturity? Under the age of 10 crime is 19 times as common among boys as among girls; the figure drops to 11 in those under 20 and five in those under 40. The biological factor, which clearly plays such a large part, must itself be complex. Much of the medical investigation of delinquency will be concerned with the problems of individual "susceptibility" to which Dr. Bowlby refers. It is probably true that tendencies to antisocial behaviour, like tendencies to neurosis, vary widely within the physiological norm, and that an actual crime is the product of a constitutional predisposition on the one hand and a situation of stress on the other. In the combating of crime, measures taken to reduce the stress factor will probably prove the most practical. Available resources would probably be used in the most productive way if some of the young hooligans whose gang activities have aroused the concern of the authorities could be captured by philanthropic agencies and have their energies directed into ways agreeable to themselves and less obnoxious to society. The removal of temptation, however, would have a wider effect than would attempts at the reform of individuals. This, however, is not really the doctor's role.

Guttmacher[2] has recently reviewed the medical aspects of crime, and his discussion shows at how many points inquiry along medical lines might prove helpful. He criticizes the impressive evidence that uniovular twins tend to be alike in criminal ways on the grounds that insufficient allowance has been made for the psychological identification which exists between uniovular twins. It is a fact that recent work has shown that Lange's original views of the overwhelming importance of genetical make-up must be modified. The hereditary factor has been found important for the development of the personality, but crime itself has been shown to be to some extent an accident. Family upbringing and traditions may prove decisive in determining whether or not a susceptible personality develops criminal tendencies. Apart from personality, intelligence too has a share in criminal predisposition: lack of intelligence makes social adjustment difficult and enhances the effect of economic stress. Most observers are agreed that on the average criminals tend to be

persons of subnormal intelligence. Mental disorder does not play a very important part except in crimes against the person. Overholser[3] has found the proportion of criminals who are insane to vary in different samples between 3% and 8%, but it is much higher among murderers. Home Office statistics for the years 1940–8 show that, of 530 persons accused of murder and not acquitted, 277, or over half, were found insane on arraignment or guilty but insane. All forms of mental disorder contribute to this total, but schizophrenia is the most important. Some mental defectives may commit murder, but their number is small. Overt and recognizable epilepsy is not specially common among murderers, but it is coming increasingly to be recognized that aggressive types of psychopathic personalities may suffer from disturbances of the electrophysiology of the brain which are akin to those of epilepsy, and that this disorder is closely associated with crimes of violence.

The psychological causes of an antisocial development of the personality cannot be neglected. There are many psychiatrists who would expect careful inquiry to disclose a disturbance of emotional relations between child and parents in the early life of most criminals. If this could be substantiated it would be to some extent a clinical confirmation of Freudian theory. Satisfactory control would, however, be essential. There are probably few people, even the most equable, who have not suffered from emotional frustrations in childhood. For the tenets of the psycho-analysts to be fully confirmed it would have to be shown that in the childhood of the criminal the psychological environment was abnormal in an unusual way or to an exceptional degree. Medical research into delinquency along these lines might be productive.

NEW CONCEPTS OF GOUT

Gout is notable not only for the distinction of its sufferers but for the elegant contributions to English literature for which it has not infrequently been responsible. Especially was this so up to the beginning of the present century. Recently Professor Wood Jones has made two such contributions, both traditional in following the observational style.[1] His Heberden Oration, which is published in the opening pages of this issue, represents the first attempt at an anatomical contribution to the understanding of gout since Sydenham wrote his classical treatise in 1683. His concept of gout as essentially a periarticular dysfunction of self-regulating arteriovenous anastomoses is convincing, for it can be correlated with many of the clinical manifestations of acute podagra and with the tendency for the disease to affect the peripheral rather than the proximal joints in accordance with the anatomical principles expressed in Hart's law.

Whatever the basic function of these precapillary arteriovenous communications (of Sucquet and Hoyer), it will be necessary to relate the changes which are now said to occur with the metabolic and endocrine abnormalities which are the concomitants not only of the acute attacks but of the disease itself. As Mason[2] has recently emphasized, gout should not be considered as primarily a metabolic disorder, for though there are obvious abnormalities of purine metabolism it has been shown that there are still more fundamental endocrine changes. In the past concentration on uric acid metabolism has proved a sterile field of research, and it was not until Wolfson and his colleagues[3] demonstrated that there was a reduction in the 17-ketosteroid excretion in gouty subjects that an advance could be made along more progressive lines. These workers also showed that such patients could, none the less, metabolize testosterone to urinary 17-ketosteroids at a normal rate after administration. Moreover, since gouty subjects do not suffer from primary or secondary sex disorder, it was postulated that an abnormal androgen is produced—the presumption being that the abnormal androgen must be produced by the adrenal gland, because both men and women can have gout.

A clue to the origin of this abnormal androgen may be provided by genetic investigations, which are rendered relatively easy by the existence of the phenomenon of hyperuricaemia as evidence of predisposition to gout. Previous familial studies tended to be disappointing until it was recognized that hyperuricaemia did not develop in affected subjects until puberty in the male or the menopause in the female. Stecher and his colleagues,[4] using serum uric acid estimations (which are said to give a sharper line of demarcation between normal and abnormal levels), found that 94% of 177 determinations in gouty subjects were over 7 mg. per 100 ml., while of 100 estimations on non-gouty subjects only 3% exceeded 6 mg. per 100 ml. They concluded that a level of 6.5 mg. per 100 ml. was evidence of hyperuricaemia in the male and 6 mg. in the female. By studies on 201 members of 44 gouty families they were able to suggest that the gene for hyperuricaemia is a non-sex-linked dominant lacking penetrance in both sexes, but with a lower penetrance in the female—this being in the neighbourhood of 84% in the heterozygous male and less than 12% in the female. These figures do offer an explanation of the well-recognized predominance of gout in the male, especially when associated with the fact that the male may develop gout at any time after puberty, while in females, as Hippocrates noted, it is rare before the menopause.

[1] *Lancet*, 1948, **1**, 165.
[2] *Med. Pr.*, 1949, **221**, 626
[3] *J. clin. Endocrinol.*, 1949, **9**, 497.
[4] *Ann. intern. Med.*, 1949, **31**, 595.
[5] *Sth. med. J.*, 1949, **42**, 720.
[6] *Science*, 1949, **109**, 280.
[7] See leading article, *British Medical Journal*, 1950, **1**, 234.

The effect of "cortisone" and ACTH on gout may be as remarkable as that on rheumatoid arthritis. Spies and Stone[5] and Hellman[6] have demonstrated the relief of the acute attack of gout by the administration of ACTH, and it has been suggested that abnormal 11-oxysteroid production may be responsible for the acute attacks, while the abnormal androgen accounts for the hyperuricaemia itself. Wolfson and his colleagues suggest that the acute attacks may in fact be due to a temporary 11-oxysteroid deficiency. Professor Wood Jones's diversion into therapeutics may not receive quite the same support from physicians who have had experience in treating clinical gout. There is evidence that ACTH, besides relieving acute attacks, may also induce gout, the attacks occurring regularly within 96 hours of its administration, at the time when the secondary rebound in 11-oxysteroid excretion, which occurs in normal individuals, fails to appear. Full doses of colchicine over this period are said to have prevented the appearance of the induced attack. Although there is still no full explanation of the mode of action of colchicine, there is some evidence that it may induce increased 11-oxysteroid production.

After more than half a century of virtual stalemate the prospects of solving the basic problems of gout are now looking brighter. Purely metabolic studies, beginning with Garrod in 1876 and since amplified by biochemical techniques in the estimation of plasma urate levels, have failed to reveal the nature of the underlying disorder. Newer concepts as yet only partially developed —such as Selye's "general adaptation syndrome,"[7] with which gout may yet be linked—at least have the function of stimulating new thought and inducing a critical outlook towards older teachings, for few diseases have more prejudice attached to them than gout. Professor Wood Jones is right to insist, as Mason has also done, on a stricter definition of gout; he emphasizes particularly the fallacy of using the term in an adjectival sense. His anatomical hypothesis should be considered with the endocrine, genetic, and biochemical hypotheses mentioned above, for it adds another piece to the jigsaw which is once more, after a period of neglect, being slowly pieced together.

A NEW BLOOD GROUP

Between the years 1900 and 1940 four genetically independent human blood-group systems were discovered, the ABO, MN, P, and Rh systems. During the subsequent decade knowledge of these has increased greatly, and four other major systems have been discovered—namely, Lutheran, Kell, Lewis, and Duffy (in addition to a number of inherited haemagglutinogens, each, as far as is known, confined to a single family). The latest to be found of this second tetrad, the "Duffy" system, was discovered and first described by Cutbush, Mollison, and Parkin.[1] It is a sign of the increasing tempo of research in this field that a second example of the antibody concerned was found within a few months and is described in this issue by Miss E. W. Ikin and Drs. A. E. Mourant and G. Plaut. In each case the discovery of the Duffy antibody was due to the application of the anti-human-globulin test of Coombs to the investigation of a transfusion reaction. The Kell antigen-antibody system was discovered by the use of the same test in the investigation of a case of haemolytic disease of the newborn, and it is probable that without this technique both these blood-group systems would have remained unknown for many years longer.

The antigen which reacts with the newly discovered antibody has been named Fy^a, and the same symbol is used for the gene which determines the presence of the antigen on the red cells. The hypothetical allele of this gene, which does not give rise to any antigen at present recognizable, is called Fy^b. Such a gene or genes are necessary to account for the inheritance of the positive and negative reactions as well as for the existence of negative reactors. It may be asked why three letters are required to describe a single antigen rather than one, as in the earlier discovered blood groups, but this development is easily seen to be inevitable. The number of known human blood-group antigens already exceeds the number of letters in the English alphabet. But the effect of the increasing complexity of the known blood groups is not simply one of arithmetic. It is essential that blood-group notation shall be acceptable to clinicians, to serologists, and to geneticists: the notation for the Duffy system has clearly been designed for these purposes. The elucidation of the Rh system, and to a lesser extent that of the ABO system, were confused by unfortunate controversies regarding notation. When there was seen to be a danger of similar confusion in the Lutheran and Lewis systems an international meeting of nearly all who had worked on these systems was held under the auspices of the World Health Organization,[2] and an agreed notation was proposed which conformed to genetical usage, which covered all the known facts, and which was capable of extension to include new discoveries. The proposed notation for the Duffy system follows a similar pattern, and there is therefore every reason why it should be universally accepted.

The discovery of a new human blood group has numerous implications for human biology, but most of them can be classed under the headings of theoretical genetics, of forensic medicine, and of clinical medicine. Recognizable genetical linkages in man have hitherto

[1] *Nature, Lond.*, 1950, **165**, 188.
[2] Andresen, P. H., Callender S. T., Fisher, R. A., Grubb, R., Morgan, W. T. J., Mourant, A. E. Pickles, M. M., and Race, R. R., ibid., 1949, **163**, 580.
[3] Chown, B., *Canad. med. Ass. J.*, 1949, **61**, 419.

been almost entirely confined to the sex chromosomes. Each newly discovered blood-group system is another potential set of chromosome markers. Thus we may hope that in the next few years blood groups and other common and readily recognizable normal characters, such as the ability to taste phenylthiocarbamide, will supply markers for most of the twenty-three pairs of autosomes in the human nucleus. Human genetics will then almost inevitably enter a new and important phase, which was reached years ago with many laboratory animals, in which linkages will be found between normal marker genes and the genes giving rise to congenital diseases. It may then at last become possible to give a precise estimate of the probability (as it already is possible for haemolytic disease of the newborn) that the child of a given couple will or will not suffer from a pathological condition already known in the relatives. When the genetics of a new blood group are firmly established and supplies of testing serum assured, it can be used for disproving paternity in affiliation cases, for the investigation of alleged accidental interchange of babies in hospital, and in attempts to identify fresh blood specimens in criminal cases. For all these purposes the Duffy system is particularly well suited, since in the general population positive and negative reactors occur in about the same numbers. On the other hand, as with all the recently discovered blood groups, the existing testing sera are all of human origin and very rare. If any of these groups are to be of use for any purpose other than research it will be necessary to find a method of preparing the antisera by the immunization of animals or, if this proves impossible, of human volunteers.

The Duffy blood groups are of little importance in clinical medicine as a whole, but in the two cases in which antibody was found they were most important. In both, the antibody was almost certainly the result of immunization by repeated transfusion. Almost certainly it was the main cause of a severe transfusion reaction in one case, and it may have contributed to the same condition in the other case. This is perhaps a negligible total of harmful effects in proportion to the enormous numbers of transfusions given since a net was thrown out to catch such cases, but a similar tale could be told of several other "rare" antibodies. In the aggregate their effects are by no means negligible, and it is certain that several more such antibodies have yet to come to light. Their formation cannot usually be avoided; it would be quite impossible to make all transfusions compatible for all the eight known blood-group systems. Certain precautions will, however, minimize the harmful effects both of the "rare" antibodies and of the rather commoner Rh antibodies. Everyone who orders or gives a transfusion should remember that blood is in many ways a dangerous substance. Transfusions should never be given lightly as a matter of convenience. A woman should not be transfused with the blood of her husband[3] or child, or indeed of any of her husband's relatives. Direct matching tests should always include a test for "incomplete" antibodies, the Coombs test for preference. This is admittedly a counsel of perfection, but in the two cases in which anti-Fy[a] was found no simpler test would have sufficed to prevent further transfusion reactions. Finally, the investigation of a transfusion reaction or of a case of haemolytic disease of the newborn should not be given up until the antibody responsible has been identified, both in the interests of the patient and in the interests of blood-group serology.

FEE FOR SIGHT TESTS

The report[1] of the Working Party on the time taken by ophthalmologists to test sight, which is summarized in the *Supplement* at page 75, shows how misguided the Minister of Health was to cut their remuneration last April. When the original fee of 31s. 6d. for a sight test was fixed, the Ministry estimated that the test would take, on the average, half an hour. Early in 1949 the Minister became disturbed about the supposedly high rate of earning of ophthalmologists, and in spite of protests by the Ophthalmic Negotiating Committee the fee was reduced on April 1 to 25s. for each sight test.

The Ministry had carried out some sort of a survey before arriving at this decision, but admitted that the information obtained was inadequate to form a sound judgment. Instead of waiting for the results of a properly conducted inquiry, it contended that three cases could be tested in an hour. The Ophthalmic Negotiating Committee argued that an estimate of 20 minutes a case was too low in view of the type of case normally referred to ophthalmologists and of the clerical work required. The Ministry compromised by fixing the fee at 25s., which is equivalent to 24 minutes a case, and agreed[2] that, "if the proposed investigation should reveal that the reduction is not fully justified, an appropriate adjustment would be made in the fee subsequently fixed."

The average time taken on sight tests is found to be 27.4 minutes—substantially more than the Ministry estimated, and barely short of the half-hour. It seems unlikely that the Ministry can quibble about the 2.6 minutes by which it falls short of 30, for the time must often be taken up by patients arriving late for the appointment, by telephone calls, and by other routine duties. There is therefore a clear case for restoring the fee to 31s. 6d., and there can be no dispute about

[1] *Report of the Working Party on the Average Time taken to test sight by Ophthalmic Medical Practitioners under the Supplementary Ophthalmic Services of the National Health Service in England, Wales, and Scotland.* H.M.S.O. 1950.
[2] *British Medical Journal, Supplement*, 1949, 1, 92.

the correction being retrospective. The representatives of the ophthalmic medical practitioners will no doubt press their claims on the Ministry in the near future.

The report includes an interesting section on eye disorders other than refractive errors found by ophthalmologists when sight-testing. The whole-time practitioners found other conditions in 40% of the completed tests, and their part-time colleagues found them in 34%. These remarkably high figures reveal what an important part the medical man plays in the eye service—a role that has occasionally been disparaged—and they emphasize the need for the provision by which patients must see their general practitioners before having their sight tested.

SPEECH IN CHILDHOOD

Since the Education Act of 1944 came into force children with defects of speech are classed as handicapped children requiring special education. Most of the research into the incidence, cause, and treatment of speech disorders in children has been carried out by isolated workers, often lacking laboratory facilities and without any special training other than their own clinical experience and professional enthusiasm. It is therefore encouraging that investigations are being conducted in Newcastle by a team consisting of a paediatrician, a psychiatrist, and a speech therapist: a report by these workers—Dr. Donald Court, Dr. Henry Miller, and Miss Muriel Morley—appears elsewhere in this issue. Perhaps in the future an otologist might be added to the team, preferably one who is interested not only in surgery but in the new science of audiology, which covers the whole field of hearing in its physical and psychological aspects.

The close relationship normally existing between a child's aptitude to acquire speech and his intellectual capacity is universally recognized, but on the other hand every paediatrician and family doctor knows that delay in the acquisition of speech is by no means invariably associated with mental backwardness. The causes for this delay are frequently obscure. It is remarkable that children first begin to recognize the significance of speech in social relationships at a very early age, usually before they have learnt to walk. The incidence of the various types of defect quoted by the Newcastle workers will be borne out by most speech therapists. The aetiology of stammering, which is the most frequent speech disability encountered among children of school age, is still uncertain. As in the treatment of enuresis, with which stammering shows many affinities, individual cases often respond brilliantly to the first treatment attempted, while others remain obstinately resistant to every known line of attack. It is difficult to believe that the factors contributing to this resistance are entirely emotional in origin. In childhood it is essential to eliminate every possible organic cause before explaining away a physical manifestation on purely psychological grounds, for otherwise, apart from the lack of relief to the individual child, there is little incentive to research.

The Newcastle workers attribute other groups of speech defects to anatomical lesions—such as cleft palate—and to auditory, neuromuscular, or cortical dysfunction. This classification, as the authors suggest, is an oversimplification, since the psycho-physiological functions of attention, memory, and discrimination are also concerned, as well as the child's stage of maturity and his opportunities for learning by experience. The problem is complex, for sounds are probably remembered in patterns rather than as a collection of individual notes, so that we recognize a tune in whatever key it is played or a word whatever vocal tone is used, just as we can read letters no matter in what size they are printed. In learning to speak a child uses all the senses except that of smell: hearing is the most important, but vision plays a definite part, especially in discriminating between the finer unvoiced consonants—s, f, th, and p. There is no doubt that the kinaesthetic memory of the organ of articulation is also involved, since it is upon the training of this faculty that the teaching of oral speech to the deaf child depends. In the education of all types of handicapped children careful study of the learning process is of great importance. The disability affects a growing organism and must therefore, in greater or less degree, influence physical, intellectual, and emotional development. In the handicapped child the acquisition of common skills can be observed in slow motion, thus providing invaluable information about the process in normal children. This is one of the reasons why the child with cerebral palsy has recently captured the interest not only of the neurologist but of the educational psychologist.

Speech in its association with language and thought is the most complicated of all human activities. As the mind matures the vocabulary increases, and the choice of words used to recall, organize, and express intellectual concepts becomes more precise. The sophisticated intelligence finds its chief delight in the contemplation of abstractions, whether in literature or mathematics. This appreciation of abstractions itself depends upon the acquisition and manipulation of the appropriate language and thus ultimately upon the child's education in correct habits of speech. A centre for the continuation of research (and perhaps for the training of medical officers) along the lines indicated by the Newcastle workers is urgently needed.

BONE AS STRUCTURAL MATERIAL

It could prove helpful, in this machine-ridden age, to determine in advance the severity of impact which the bones of any individual might be expected to withstand undamaged, or, in the language of the engineer, to determine the breaking stress of a bone without "testing to destruction." We have already commented on one line of research conducted by the U.S. Bureau of Standards[1]: from accurate measurements on a rather limited number of bones in compression (as in the leg bones on landing from a jump) it was concluded that there was a good correlation between the density of healthy bone and the extent of violence which it can tolerate, and the suggestion was made that bones of abnormal weakness might be detected radiographically. Professor G. H. Bell and his colleagues in Scotland have

[1] *British Medical Journal*, 1949, 1, 449.
[2] *J. Bone Jt Surg.*, 1949, 31B, 444.

been interested since 1941 in the testing of bones under different conditions, and they have lately published an investigation on the strength and elasticity of the bones of rats fed on one or other of three diets.[2] This suggests that a useful indication of the "goodness" of living bone can be obtained more directly. The method of test was to suspend an intact femur from its two ends, attach a series of weights to its mid-point, and record the deflection produced. This, it is pointed out, "reproduces experimentally one of the strains to which living bones may be subjected by loads or muscular action." It is also an easier measurement to make practically than that adopted by the American investigators. From a series of such measurements the elastic constant of the bone—as normally defined—can be readily determined. Further, if testing is continued until the bone breaks, there is a good correlation on the average between breaking stress and the elastic constant. As an indication of scale, the breaking stress for a group of rats fed on the Rowett Institute stock diet, and therefore adequately nourished, was 26,900 lb. per sq. in. (1,880 kg. per sq. cm.); that for rats fed on a rachitogenic diet plus vitamin D was 18,900 lb. per sq. in. (1,163 kg. per sq. cm.); and that for rats fed on the same diet, but without the vitamin, was 11,900 lb. per sq. in. (830 kg. per sq. cm.). If such figures can be regarded as typical of the more serious departures from normal, then an indication of quite moderate accuracy could at times be useful: hence Professor Bell's suggestion that "useful information might be gained by measuring the elasticity of living human bones." In any case, the connexion between breaking stress and elastic constant is closer than that between breaking stress and mineral content—shown by percentage of ash on ignition—so that on the evidence presented this should prove the more hopeful of the two procedures.

TERMINAL CARDIOGRAMS

Electrocardiograms show a wide variety of rhythms and patterns shortly before death and for a varying number of minutes after signs of cardiac activity have ceased. Massie and his colleagues[1] have studied tracings taken at these times in 34 cases. They found that within half an hour of death there is depression of the centres of stimulus production, with sinus bradycardia, sino-auricular block, A–V nodal rhythm, and various grades of A–V heart block. At the same time there is a change in the properties of the contracting tissues, resulting in extrasystoles—both auricular and ventricular—and ventricular tachycardia, flutter, and fibrillation. Conduction may be depressed through either of the bundle branches, and either ventricle or auricle may continue to show some activity after the other has ceased. Auricular fibrillation never appeared as a terminal event. In 18 cases the effect of intracardiac adrenaline (1–2 ml. of 1 in 10,000 solution) was observed after the cardiogram had ceased to record any cardiac activity. There was some sort of response in seven, up to a maximum of 36 minutes, though in only two was it normal sinus rhythm. Adrenaline more often caused a response when injected into the myocardium than when injected into the cardiac cavities.

[1] *Ann. Intern. Med.*, 1948, **29**, 838.

SPONTANEOUS PERFORATION OF THE OESOPHAGUS

The signs and symptoms of spontaneous perforation of the oesophagus are so characteristic that it is surprising that the entity has only recently been recognized. Formerly this was a condition almost always rapidly fatal, but since thoracotomy has become an everyday procedure a few patients have recovered, and early diagnosis is now of the greatest importance. Several case reports have recently been published, the total number of recorded cases amounting to nearly seventy. The rupture is always a vertical tear of the lower end of the oesophagus. Scholefield[1] stresses the fact that the remainder of the organ is normal, but in three of the cases described by Kinsella and his colleagues[2] oesophagitis or ulceration was present: the histories suggest that these three patients had symptomless peptic ulceration of the oesophagus. A similar longitudinal tear occurs in its lower third when the oesophagus in the cadaver is distended by air at an average pressure of 190 mm. of mercury. An unusual case has recently been described by Kennard[3] in this *Journal*, the rupture occurring during childbirth. The patients are usually men, middle-aged or old, and often alcoholic or gluttonous. The symptoms occur after vomiting: excruciating epigastric pain has a rapid onset and spreads to the lower chest and back. It is not relieved by morphine. The patient becomes shocked and usually dyspnoeic and cyanosed, with low temperature and rapid pulse and respiration; he prefers to sit up, and complains of thirst. At first there is epigastric tenderness and rigidity, and perforated peptic ulcer may be strongly suspected. But before long two characteristic phenomena develop: a pleural effusion, usually with associated pneumothorax, and, very frequently, subcutaneous emphysema, spreading from the posterior mediastinum to the neck and first seen over the upper part of the sternum. *X*-ray examination provides confirmation. The earliest sign is loss of translucency behind the heart, due to air escaping into the mediastinal tissues; this spreads, and a fluid level appears. A hydropneumothorax may be seen, but there is no free air below the diaphragm. Barium or iodized oil when swallowed passes into the pleural cavity. Finally, gastric contents can be aspirated from the pleura.

Without surgical treatment the prognosis is grave, and only a very few spontaneous recoveries have been reported. Moore and Murphy's[4] patient, a man of 33, recovered after thoracotomy on the twenty-first day. The authors advise drainage of the chest and jejunostomy and consider early thoracotomy unwise, but here they differ from the other authors, who recommend closure of the perforation as soon as essential resuscitation has been started. Immediate operation was successful in Scholefield's case, a woman of 63 who was *in extremis*. Most surgeons agree that early operation is the best treatment, and that the essential requirement to save these patients' lives is early diagnosis. Although the condition is rare, it should immediately come to mind if the unusual syndrome is encountered of severe pain after vomiting, together with hydropneumothorax or subcutaneous emphysema.

[1] *British Medical Journal*, 1949, **1**, 348.
[2] *J. thorac. Surg.*, 1948, **17**, 613.
[3] *British Medical Journal*, 1950, **1**, 417.
[4] Ibid., 1948, **17**, 632.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

ACUTE ABDOMINAL DISEASE

BY

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In acute abdominal disease, diagnosis—early diagnosis—is the difficult part of the case: when the correct diagnosis has been made the treatment is not usually in doubt. The practitioner frequently has to make important decisions in diagnosis quickly, and often without anything but his clinical acumen to act as guide. In hospital, and occasionally elsewhere, he may be helped by the laboratory or the radiograph, but in general the life of the patient will depend upon his careful observation and wise judgment.

The first fact to determine is whether the cause of the pain lies within the abdomen or whether it is referred from a lesion in the pleura, the lung, the heart, the spinal column, or the spinal cord. As a rule, the paroxysmal or intermittent nature of the pain, the extent of tenderness and rigidity of the abdomen, or the easily ascertained deep tenderness will quickly enable one to decide that the abdomen contains the cause of the trouble. But it cannot be too strongly emphasized that in every case the chest must be examined before a final diagnosis is made. Moreover, it should be a routine to test the knee-jerks and the reaction of the pupils to light and accommodation, for the gastric crises of tabes have often deceived the observer into thinking that there was a surgical cause for the abdominal pain. The pre-eruptive stage of a lower dorsal herpes has also on occasion simulated an abdominal lesion, but the extreme hyperaesthesia over the area of distribution of the affected nerve should put one on guard.

Many, if not most, abdominal catastrophes occur in patients who have previously felt quite well. If there has been previous malaise or ill-health one must remember that there may be some irregular abdominal pain with such general diseases as influenza or typhoid fever, and that too much or too little sugar in the blood of a diabetic may bring about a state in which there may be abdominal pain, vomiting, and sometimes abdominal rigidity.

Abdominal pain may occur alone or may be accompanied by vomiting, rigidity of the abdominal wall, or abdominal distension. The six main types of abdominal emergency are the colics, perforations, inflammations, obstructions of the intestine, torsions of the viscera, and vascular lesions.

The Colics

Severe paroxysmal or colicky pain occurring without distension or rigidity is usually due to one of the colics. Pain in one or other loin radiating to the groin and causing the patient to writhe and retch, if accompanied by tenderness in the erector-costal angle, is almost always due to renal colic. Tenderness in the same spot, with a turbid urine, fever (up to 103° F.=39.4° C.), and perhaps frequency of urination should be due to pyelitis. Biliary colic may give rise to a girdle pain or may be strictly localized in the right subcostal and subscapular regions; it is often very severe, but rigidity is not present unless there is accompanying cholecystitis. Uterine colic is chiefly felt in the hypogastric and sacro-lumbar regions.

Intestinal colic, commonly due to indiscretions of diet, is felt chiefly in the midline anteriorly, small-gut pain above or round the navel, and large-gut pain in the hypogastrium and sometimes in the small of the back. Appendicular colic, and the early pain of acute appendicitis, is usually felt in the same position as small-gut colic, and does not cause pain in the right iliac region until the inflammation comes through the appendix wall and irritates the parietal tissues in the iliac fossa. Any case of doubtful intestinal colic should therefore be seen again within a few hours, when more localizing signs should have developed if the case is one of appendicitis.

The Perforations

Rigidity of the abdominal wall is the most characteristic feature of peritonitis, which is rarely primary but more often follows the perforation or inflammation of a viscus. There is one form of peritonitis in which there may not be any abdominal rigidity—pneumococcal peritonitis. This occurs in girls between 5 and 10 years of age and causes abdominal pain and tumidity (chiefly in the hypogastrium), but there are also general symptoms and sometimes delirium, and one may be able to obtain a positive culture of the pneumococcus from the blood.

Perforation of a gastric or duodenal ulcer, or of any part of the small or large intestine, may be accompanied by initial collapse, but is chiefly diagnosed by the immediate, extensive, and persistent rigidity and tenderness of the abdominal wall. If the pelvis be involved there will be tenderness of the pelvic peritoneum as felt per rectum. If a radiograph can be taken some free gas may be revealed between the liver and the diaphragm, and if there is a large amount the liver dullness in the axillary line may be diminished.

The Inflammations

Inflammations of the appendix, the gall-bladder, Meckel's diverticulum, the Fallopian tubes, or the pancreas usually start with abdominal pain, followed by slight vomiting without any change in the tension of the abdominal wall. As the inflammation extends fever develops and localized rigidity of the abdominal wall appears in the region of the viscus. But such rigidity in any region may be caused by any one of several conditions. In the right upper quadrant it may be caused by cholecystitis or a slowly leaking duodenal ulcer. In the right lower quadrant the most common cause is appendicitis, but every experienced practitioner knows that rigidity in this region may also be caused by an inflamed Meckel's diverticulum, by regional ileitis, by a leaking duodenal ulcer, by acute pancreatitis, or, more rarely, by inflamed glands. Rigidity in the left iliac region is most commonly due to diverticulitis of the sigmoid colon. General hypogastric rigidity may be due to peritonitis ascending from the pelvis, caused by salpingitis or a ruptured pelvic appendix. A strangulated coil of gut causes local peritonitis and overlying muscular rigidity.

Obstructions of the Intestine

Apart from the case of a strangulated coil of gut with local peritonitis intestinal obstruction does not cause abdominal rigidity. It is recognized by the occurrence of recurring bouts of acute central abdominal pain accompanied by frequent vomiting if the obstruction is in the jejunum, less frequent vomiting when the stoppage is in the ileum, and often with little or no vomiting when the large bowel is obstructed. After the lower bowel has been emptied by enema no gas or faeces pass per rectum. Sometimes peristalsis may be visible through the abdominal wall. In every case of suspected intestinal obstruction the first thing to be done is to examine the hernial orifices, as a strangulated hernia is the commonest cause. A small femoral hernia the size of a filbert may be the unobserved cause of the obstruction.

Intussusception in infants is a common cause of acute abdominal pain and, usually, obstruction. Intermittent screaming attacks with drawing up of the legs, a sausage-shaped tumour in the upper part or left side of the abdomen, and later the passage of blood and mucus per rectum point to intussusception; the apex of the invaginated gut may sometimes be felt per rectum. A radiograph after a barium enema will show the intussusception.

Volvulus of the sigmoid causes speedy distension of the abdomen. Vomiting does not usually occur, and the symptoms are often subacute and give time for a plain radiograph to be taken, in which the distended coil can be clearly seen. Radiographs are also most useful in other cases of small-gut and large-gut obstruction. Fluid levels in the small bowel and sharply cut division between a length of gas-distended colon and an empty succeeding portion point to obstruction at the junction between the two parts.

Torsions

Torsions of viscera cause severe pain, vomiting, local tenderness, and swelling, leading to rigidity as peritonitis supervenes. In the female a twisted ovarian cyst and fibroid tumour must be kept in mind. Rarely the gall-bladder and very rarely the stomach can be twisted on their axes. A twisted portion of omentum may thrombose and become necrotic, causing severe localized pain.

It is seldom that torsions are diagnosed prior to operation.

Vascular Lesions

The vascular lesions which may cause serious abdominal pain are mesenteric thrombosis and dissecting aneurysm. A dissecting aneurysm may cause agonizing abdominal pain and rigidity of the abdominal wall, but the pain is first felt in the thorax (and sometimes in the left arm) and gradually descends to the abdomen. Mesenteric thrombosis may cause intense central abdominal pain, and as the involved gut becomes infarcted an oval swelling may be felt in the centre of the abdomen; blood may be passed per rectum or be seen in the result of an enema.

The examination of an abdominal emergency is not complete until a rectal examination has been made and the urine tested for protein and sugar. The picture of uraemia may easily look like that of intestinal obstruction.

Treatment

The treatment of some forms of acute abdominal disease has undergone modification during the past few years. This particularly applies to the acute inflammations and intestinal obstruction.

Perforation of any part of the stomach or intestinal canal needs prompt exploration to enable the opening to be closed. It is true that recently several surgeons have proved that conservative treatment of a perforated peptic ulcer can be successful. They have shown (and I have confirmed) that by keeping the stomach empty (by indwelling tube and by withholding all fluids by mouth) the perforation may be sealed off and the peritonitis may subside. Yet this method should be reserved only for special cases—for desperate cases in which immediate operation would almost certainly kill the patient or for patients seen in remote parts where surgical help is not available.

Of the various inflammations gonococcal salpingitis and pneumococcal peritonitis should be curable by the administration of penicillin. Half to one million units daily should suffice, given in concentrated form intramuscularly.

Acute cholecystitis seldom causes such dangerous spreading peritonitis as does appendicitis, and many surgeons prefer if possible to allow the acute stage to subside under conservative treatment and to remove the gall-bladder later. If the gall-bladder necroses it will, of course, be necessary to drain any local abscess which forms.

Acute pancreatitis, which formerly was treated by immediate operation, is usually treated conservatively, for experience has shown that there is little benefit from surgery. Later in the course of the disease it may be necessary to drain a pancreatic abscess. When gall-stones are thought to be present some surgeons prefer to operate, remove the stones, and relieve tension in the biliary ducts by draining the gall-bladder. Unfortunately it is not easy to make sure of the diagnosis of acute pancreatitis without laboratory aid to determine the amount of diastase in the urine. When the urinary diastase is much raised it clinches the diagnosis of pancreatitis.

Appendicitis

The results of treatment of appendicitis are still not so good as they should be. Everyone agrees that whenever possible the appendix should be removed within the first 48 hours of the attack. After that time has elapsed in very acute cases the presumption is that the appendix has perforated, and surgeons differ regarding the best method of treatment. Some still advise immediate operation as soon as possible after the case has been seen, in spite of the fact that this sometimes leads to the spread of an infection that might have spontaneously localized. Others consider it better to follow conservative treatment with a view to allowing the inflammation to subside so that appendicectomy may be done later in the "cold" state. This knotty question cannot be discussed here. Suffice it to say that if conservative treatment be adopted it must be conscientiously carried out. The patient must be kept in bed under skilled supervision, accurate records of the pulse and temperature must be taken, and vomiting and increase of pain should be regarded as serious danger signals indicating the need for intervention. The stomach should be emptied by aspiration through a Ryle's or similar tube, and bowel immobility must be procured by allowing nothing to be taken by mouth for 24 to 48 hours, and thereafter fluids must be given in small quantities gradually increasing. Aperients and enemata must be avoided. The general state of the patient should be maintained by parenteral administration of fluids, pain should be quelled by small doses of morphine, and infection should be combated by injections of sulphonamides or penicillin. It is probably better to operate promptly in all but two special types of case. The first is the patient with advanced general peritonitis who is in such a poor state

that any operation would probably prove fatal; such a patient may benefit by complete rest, by injections of sulphonamides and penicillin, and by fluids given intravenously, rectally, or subcutaneously. In a few days the abdomen may be drained by making a small incision under local analgesia. As a rule vomiting soon stops and feeding by the mouth can then start. The second type of case is that in which the surgeon first sees the patient after the formation of a well-defined and localized lump which indicates that the natural resources are walling off the infection. Usually this lump gets less tender and gradually diminishes under conservative treatment. If it shows a tendency to increase or if unfavourable symptoms appear there should then be no hesitation in operating at once.

Intestinal Obstruction

The treatment of intestinal obstruction was, until a few years ago, almost always immediate operation, and the results were very bad. It is now recognized that it is of the utmost importance to improve the patient's general condition before operating. Dehydration must be combated by the administration of fluid parenterally, and toxaemia must be reduced by passing a small rubber tube into the stomach and removing the noxious contents. The tube should be kept in the stomach during operation so that there should be no risk of aspiration of septic contents into the lungs.

Everyone agrees that operation should be prompt when gut is strangled, but in cases of obturation, or obstruction by a kink or adhesion, Wangensteen has shown that the distension can be diminished and sometimes the obstruction relieved by means of the Miller–Abbott tube. This tube is swallowed and the stomach emptied by suction through its lumen; the tip of the tube is then allowed to pass through the pylorus into the duodenum and then into the jejunum, while suction is maintained through the tube. Even if the obstruction is not relieved the reduction of distension makes operation safer and more easy. This method should not be continued if little or no improvement takes place and should never be undertaken unless strangulation can be excluded.

Intussusception can sometimes be relieved without operation by giving a barium enema under low pressure. The child's abdomen must be carefully watched under the fluorescent screen, and complete reduction can be certified if the barium passes back into the ileum through the ileo-caecal valve. If there is the slightest doubt about the reduction operation should be performed to make certain and to complete reduction if necessary. For this reason the barium injection should be given only after the child has been prepared for operation, and the theatre should be kept ready to proceed with the operation without delay.

Mesenteric thrombosis sometimes demands exploration and may require resection of the involved portion of intestine. Nothing can be done for a dissecting aneurysm.

Ectopic gestation is an emergency which does not come into any of the preceding categories. When a woman of child-bearing age misses a period, complains of lower abdominal colicky pain, perhaps has a little irregular uterine bleeding, and if on examination a rounded tender lump is felt on one side of the uterus, then one must suspect a tubal gestation. If in a woman with such a history the signs of intra-abdominal haemorrhage suddenly develop, then one must conclude that the ectopic gestation sac has ruptured and needs prompt operation.

Conclusions

To sum up: there have been few new diagnostic aids to assist the practitioner to unravel a puzzling acute abdominal emergency; the main improvement has come from the more general use of radiographs, which greatly assist in the diagnosis of many forms of intestinal obstruction and in some cases of perforated ulcer.

In treatment there has been considerable improvement in the case of peritonitis and the various inflammations by the judicious use of the sulphonamides and penicillin: and the greater care in the pre-operative preparation of patients suffering from intestinal obstruction has been of great benefit. Moreover, the post-operative use of penicillin in cases of peritonitis or after bowel resection has improved the results.

THE GENERAL PRACTITIONER AND THE MATERNITY SERVICE*

BY

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I need not remind you that this subject has been responsible for more difference of opinion between the general practitioners and the specialists than any other section of the Health Act. Many people have said that the Health Act will put back the clock of midwifery by 20 years, but I do not think that this is true. The Act has given us a skeleton service, and it is up to us to clothe it. In spite of the chaotic regulations that exist at present, I think that it is capable of being moulded into something better than we have yet seen. But this is certain to take time, like any other process of evolution, and we must beware of the administrative pen that would bring about these changes at one stroke. First we must decide what we want, then we must adapt it in some practical form, and finally we must prove that it will work.

Maternal Mortality

In order to see where we stand to-day we must have some idea of the maternal mortality rate for the last 15 years. The simple diagrams that I have prepared should be perfectly clear to everyone. They are to be regarded as a general impression, and are accurate enough for our purpose.

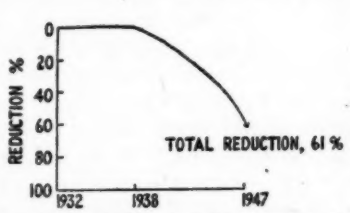


FIG. 1.—Showing the decrease in the maternal mortality rate, 1932–47.

Fig. 1 represents the fall in maternal mortality during the period 1932–47. At first sight this is a satisfactory drop, and various explanations have been given for it. Some obstetricians would stress better obstetric management as the chief factor, others that chemotherapy has played the greatest part, while politicians are inclined to emphasize new policy by the Government. But let us see what an analysis of the figures really shows.

*The chairman's inaugural address (abridged) to the East Herts Division of the British Medical Association.

In Fig. 2 I have separated the causes of maternal mortality into four groups: sepsis, toxaemia, haemorrhage, and a fourth group containing all the other accidents of childbirth not covered by the first three. The average of these four groups is of course represented by the overall mortality in Fig. 1.

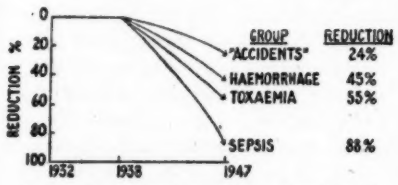


FIG. 2.—Showing the reduction in maternal mortality from different causes, 1932–47.

It will be seen that the highly satisfactory drop in deaths from sepsis began with the general use of chemotherapy in 1938 and has now reached the low reduction figure of 88%. The fall in haemorrhage cases by 45% has been brought about chiefly by the greater availability of blood transfusion. No doubt improved obstetric methods have played some part, but it is a fair comment to point out that the improvement in these two groups has been brought about largely by the application of discoveries in general medicine to our obstetric cases.

In the toxaemia group the position is different, and it can certainly be claimed that the reduction by 55% of deaths from this group of diseases is almost entirely due to better antenatal care.

The fourth group of "other accidents of childbirth" shows the least reduction of all. Deaths from these causes have fallen by only 24% in the 15 years, and it is this group with which we should be most concerned, for it represents to a large extent the standard we have reached in the actual practice of obstetrics.

History of the Present Situation

It cannot be said that there was any real attempt at prevention of difficulty by organized antenatal care until the first Maternity and Child Welfare Act of 1918. This was good as a start, but it regarded antenatal care as a separate thing, carried out by the clinic doctor, who had little contact with the midwife who delivered the case. If the midwife required help she sent for a general practitioner who had never seen the case, and no post-natal work was done.

So things went on until 1932, when the Ministry of Health, being somewhat perturbed at the high maternal mortality of over 4 per 1,000, published its first report. At the end of the report this brief comment was made: "46% of these deaths were avoidable."

By 1937 there was still no fall in the maternal mortality, and the Ministry issued a second report, this time adding what might be called "the first hint of things to come." At the end of the report it was stated: "It is only those practitioners who show special interest in and have special experience in midwifery who should be called to the assistance of the midwife."

Ideas of a health service were now beginning to take shape, and the British Medical Association published a "Health Service for the Nation." In the maternity section three things were suggested: (1) antenatal care should be carried out by a practitioner chosen by the patient; (2) a certified midwife should attend all cases; and (3) attendance by the same practitioner during labour when he or the midwife considered it necessary.

Then came the war, and all planning ceased. But during the war some interesting things happened. The Emergency Medical Service was born, and with it the Emergency Maternity Service. In Hertfordshire we became very familiar with this, and we were fortunate in having first Sir Eardley Holland and later Mr. F. Neon Reynolds to help us in the organization of this service. I believe a great deal was learnt at this time that has subsequently been of use in planning the new maternity service.

After the acceptance of the Beveridge Plan and the first White Paper we began to see the first heavy ammunition against the general practitioner. In 1944 the Royal College of Obstetricians and Gynaecologists published a report on the maternity service. It stressed the advisability of special experience for general practitioners undertaking maternity work, and it is worth while quoting one part of the report with which I am sure no one will quarrel. It said: "The present system often places general practitioners in impossible situations. Unaided and in unsuitable surroundings they may have to deal with complications that would test the skill and promptness of the most eminent specialist. General practitioners and their patients should be protected by a maternity service that makes provision for such situations."

This was followed by a similar report from Northern Ireland and by the Goodenough Report on Medical Schools. The latter went so far as to say that general practitioners should not deal with abnormal midwifery. And so the great dispute between general practitioners and the obstetric specialists began to take shape.

Some Reflections upon the Emergency Maternity Service

Now at this time, in 1944, I was in the rather unusual position of running a large country maternity practice and also an obstetric hospital service of about 100 beds, and I had the unique opportunity of being able to examine both sides of the question at the same time.

I thought of the good and the bad sides of general-practice midwifery. If it goes well it is all right, but if it does not it can be frightful. All of you must have had similar experiences in general practice: the unexpected presentations, the large breech in a small pelvis, the cases of inertia, the time factor, and even the simple forceps delivery that goes wrong. Perhaps you are just about to turn a simple posterior position. You have handed over the chloroform to the district nurse, when suddenly there is silence. You look at the other end, and there are the enormous pupils and the grey face. You drop everything and give artificial respiration. The patient starts breathing again, but you are now thoroughly frightened and you pull the baby out hurriedly face to pubes. She is not really fit enough for a proper repair, so you put a couple of silkworm sutures "right through everything." Just as you think that all is well there is the dreaded sound like water dripping from a broken gutter. There is blood everywhere, on the floor, on the walls, and on your trousers, but somehow the patient survives. It is a frightful picture—and so is the perineum.

These are the cases that we should all like to get rid of if we could. But what should we like to preserve? First, I think, the interest. Many general practitioners are extremely interested in midwifery and it would be a pity to take it all away from them, because they are the ones who become very good at it. Secondly, maternity work produces a bond with the family that nothing can replace in general practice. It will be regrettable if we lose this bond, although I fear that it may not be possible to preserve it as we have known it in the past. Thirdly, I think we want to preserve some midwifery for the general

practitioner from the point of view of the Service, because he is the first line of defence.

You may perhaps be called out at 3 a.m. because a patient you have known for years has abdominal pain. You do not know that she is seven months pregnant and has been attending a clinic. You are not considered to be good enough to do her antenatal work, but you are left in the middle of the night to sort out all the causes of abdominal pain in pregnancy. You probably feel like telling her to send for the clinic doctor—only he would not be on the telephone.

Having thought on these lines in 1944, I came to the following conclusions: If general-practitioner midwifery is to survive the criticism that is being hurled at it general practitioners must realize three things: (1) they must learn to do better antenatal work; (2) they must be helped by a better hospital service; and (3) they must learn to use this help at the right time.

When Sir Eardley Holland left Hertfordshire, where he had come to know many of the general practitioners, he wrote an article in the *Practitioner* in which he said that he thought the practitioners themselves would make the change from within. The change is certainly coming; but I believe now, as I did then, that it will never come from within. It will have to come from without, through the hospital service.

Conditions at the Present Time

We are now in a position to consider the present situation. What has the Act given us? It has given us three separately controlled departments in obstetrics. The obstetricians are under the regional hospital boards, the general practitioners are under the executive councils, and the midwives are under the local authorities. There is at present no link between them, and if we consider the position of each separately it will be obvious that this administrative blunder strikes at the very root of the service. It is surely a case of "United we stand, divided we fall."

The maternity hospitals are in a fairly satisfactory position. We are left with considerable freedom to organize ourselves locally, although we have our troubles. There is still a great reluctance to send in cases early enough, although the recent decision by the Ministry that practitioners may claim the full fee where a case is transferred to hospital during labour should improve this situation. Most obstetric calamities arrive at the maternity hospital in the end. There is no excuse left for dealing with these cases "unaided and in unsuitable surroundings."

You will tell me, of course, that you lose touch with your patients in hospital, that often they come home with a breast abscess or a piece of placenta left behind. Your babies come out uncircumcised and with "sticky eyes." We are only too well aware of this, and, although I suspect that you make the most of these cases, it is one of our greatest problems. I should like to see our "flying squads" extended to follow up some of these post-natal complications. Nevertheless, in the maternity hospitals the framework of a good service is there already.

General practitioners are so well aware of their own position as it affects themselves that I shall only briefly mention it. In spite of the good intentions of the Act, the regulations have now been amended so that every general practitioner is regarded as an obstetrician. There is nothing whatever in the Act to prevent you doing all the midwifery in connexion with your own cases, and it is interesting to note that the Ministry values extra experience at precisely two guineas. And so we arrive at the creation of the "seven-guinea practitioner."

The midwives are perhaps in the most uncertain position of all. It is said that in 1947 86% of all births were attended by midwives; but now the responsibility often passes to the general practitioner and they become maternity nurses, although they may still have to deliver the case by themselves. They resent this and feel frustrated, so that the end-result may well be an even worse shortage of midwives. They are also dissatisfied at referring some of their cases to the general-practitioner obstetrician. Only the other day a midwife told me that she had sent one of her cases to a doctor. The patient had some oedema, albuminuria, and a blood pressure of 160/100. The doctor told her not to worry; it would be all right. But they do worry, and they do not like it.

Another midwife received a note from a doctor about one of her cases. The doctor had diagnosed disproportion in a primipara and the head was above the brim. He asked the midwife to give the patient castor oil at home, and he enclosed some oestrogen pessaries, which he suggested should be inserted into the cervix. He hoped that this would start labour, but if the head did not come down after three hours in the second stage he hoped that she would send for him. I cannot help hoping that the patient is still alive.

These stories may sound fantastic; but evidence of this sort is sifted and collected by the various authorities, and does not help the position of the general practitioner, for the good suffer for the bad.

So all three groups are unhappy: the specialists because they do not see the cases early enough and they have no link with the rest of the service; the midwives because they feel that the doctors are taking away their job, which they could do better themselves—let us admit that—and because they do not altogether trust the "seven-guinea obstetrician"; the general practitioner because he is being criticized by everyone and feels that he is being pushed out of yet another part of general practice, with loss of interest and income. That is briefly the present position.

Steps Taken to Improve Matters

So far very little has been done to sort out the muddle. On August 6, 1949, the Maternity and Midwifery Advisory Committee of the Central Health Services Council published a statement to "clarify the position." This statement has been criticized so many times that I will only refer to it in passing. But it should be remembered that it was prepared not by the Ministry but by a committee upon which many obstetricians were present. We must give the Ministry its due that it published the statement.

The full statement appears in the *Supplement* to the *British Medical Journal* of August 6, 1949 (p. 74). "Misunderstanding exists," it began. When a "doctor undertakes to provide maternity medical services" under the Act *it is not the intention of the Act that he "accepts full responsibility"* (italics mine). Then we go on to hear about teamwork all over again. And who is to be responsible for the patient? How is the team to work together? And who is to be responsible for the team?

You will not expect me to solve all these problems here and now, but so far we have had no answer from the planning bodies. It is of course quite easy to pull the whole structure to pieces, and it is only fair that I should be asked to put it together again. Unfortunately we cannot at present alter the pieces, but they can be arranged differently and I think more effectively.

What Lies Ahead?

There are those who would do away with domiciliary midwifery altogether. The number of hospital beds available and the total number of confinements would just allow five days in hospital for each case. I think that this is an undesirable and an unlikely solution. We must assume that domiciliary midwifery will always exist, and if social conditions improve it may even increase. It would certainly increase if the hospitals made a small charge for each confinement.

Our first problem is obviously one of integration. We can make no progress until there is one control of all midwifery. This I think should be regional rather than for the whole country, for a service suitable for London would not work in Cumberland. The next step is to form a committee to work out the details, and this committee should be made up of obstetric specialists, general practitioners, midwives, and the local authorities.

In domiciliary midwifery we must look ahead, perhaps 20 years, to see what we are aiming at. By then we should have evolved the true general-practitioner obstetrician in sufficient numbers. He would possess a diploma, or at least would have held posts in maternity hospitals so that he would be familiar with the other branch of the service. I would then make the domiciliary service the responsibility of the general-practitioner obstetrician, who would himself be responsible to the maternity hospital.

Antenatal examinations for domiciliary cases would be carried out at the health centre (if these are in existence in 20 years) by the general-practitioner obstetrician, and the midwives would attend with their own cases and take over some of the examinations. In normal cases the midwife would be responsible for the delivery, and if difficulty appeared she would send for the general-practitioner obstetrician who had seen the case at the health centre. This would mean that the local authority clinics, if they continued to exist at all, would change in character. Obstetric examinations would no longer be carried out at them, but they could be useful in providing health talks, demonstrations upon relaxation, and instructions about diet, analgesia, and other subjects of interest to the expectant mother.

But we must return to earth and to the present. We have no health centres, and all general practitioners are obstetricians, therefore the immediate solution must be a compromise. We cannot rely upon the minimum antenatal examinations provided for by the Act, or upon such examinations as at present carried out by general practitioners. Somewhere in the region of seven million antenatal examinations are carried out yearly by the various clinics. The general practitioners could not attempt to take over this work entirely, and therefore, for the present, the local authority clinics and midwives must continue to carry out the intermediate examinations where the doctor lacks either the time or the interest to do so.

We can rely upon the midwives, for they are both keen and conscientious, and, moreover, they are answerable for everything that they do to the Central Midwives Board—a fact that has largely contributed to their efficiency. The general practitioners are answerable to no one, so we are left to find some means of improving the obstetric experience of all those who are for the present to be allowed to continue as consultants to the midwives.

First, I would suggest postgraduate demonstrations and discussions at the nearest maternity hospital. This would provide an opportunity for reviewing recent advances in obstetrics, discussing common problems such as toxaemia and ante-partum haemorrhage, and stressing the procedure for admitting these cases to hospital. For example, very few cases of ante-partum haemorrhage are admitted to hospital without a note from the doctor stating that he has felt a "boggy mass" high up in the vagina and that he thinks it must be a case of placenta praevia. Possibly it is, but the boggy mass is more likely to be blood clot, and no one can be sure where the placenta is situated until that patient is examined properly under an anaesthetic. Caesarean section is now used a great deal in the treatment of placenta praevia, but the mortality rises with each vaginal examination that has been made before delivery. At these discussions it could be explained why it is wiser to send such a case into hospital without making a vaginal examination.

There is only one criticism of these postgraduate demonstrations. Only those practitioners who are interested in midwifery would come. It is the others with whom we really need to make contact.

In North Hertfordshire we have been trying out another method by which the general practitioner can be kept in touch with the maternity hospital. It concerns the antenatal care of patients who are to be delivered in hospital. Many of them have to travel long distances, and in these cases our procedure is as follows. The patient must attend the hospital for booking and the first examination. A complete overhaul is made and blood tests are taken. If no abnormality is found we ask the patient's own doctor to look after her until the 36th week, when the hospital again takes over the examinations. This means perhaps six antenatal examinations by the general practitioner, for which he can claim three and a half guineas under Part I of the executive council's scheme.

If any abnormality is found at the first examination, such as mitral stenosis or chronic hypertension, then we see the case throughout pregnancy. Others may be asked to return earlier than the 36th week, as, for example, when an Rh-negative mother has been found to have antibodies present in the first test. We trust the practitioners to refer any case back to hospital if any abnormality appears, and so far this system has worked extremely well. It seems to be appreciated by those practitioners who are interested, for they are kept in touch with their own cases and also with the standards of the maternity hospital.

I believe that this system could be extended to more of our hospital cases, and the pressure on our clinics would be relieved in this way, with benefit both to the patient and to the general practitioner. One soon learns which practitioners can be trusted to do reliable antenatal work, though I would not like to suggest that we practise an unofficial selection.

The Stillbirth Rate.—So far we have not considered the question of stillbirths. There has been a steady fall in the stillbirth rate for the last 10 years. It is now 23 per 1,000, and there has been no great improvement lately, although each year still shows a gradual reduction. I think that we should review all cases of stillbirth. It is done in America, it is done in our hospitals, and if a midwife notifies a stillbirth there is a full inquiry. Why should this not be extended to general practitioners? It might be resented at first, but it would lead to better work in the end.

The First Year of the Act

I have already hinted that I do not fear the "putting back of the obstetric clock," nor do I see in imagination hordes of general practitioners swarming into the field of obstetrics. We have seen only the first year of the Act,

but the figures available for Hertfordshire show that as yet there has been very little difference in the ratio of doctor–midwife deliveries. Dr. J. L. Dunlop, our county medical officer, has kindly supplied me with the figures for the six months immediately preceding the Act and for the first six months of 1949. These figures show only a 4% increase in doctors' cases, and this should help to reassure our midwives that their work is not being taken away from them.

Nevertheless, we have not yet seen the full results of increased general-practitioner midwifery, and I suggest that you watch the future maternal mortality very carefully. If there is an increase in mortality then general-practitioner midwifery is finished for all time, for this is just the piece of evidence that is awaited by the authorities.

The maternal mortality rate for Hertfordshire for the last two years has reached the very low figure of 0.5 per 1,000, and to maintain and improve this figure will need the co-operation of everyone responsible for maternity work.

The Need for Further Progress

One final glance at the maternal mortality figures should convince everyone that further progress is possible. Even at the lowest point in 1947 toxaemia of pregnancy accounted for 239 deaths, and the "other accidents of childbirth" for 206. One baby out of every 25 does not survive the hazards of labour and the neonatal period.

We must accept the amended regulations for the domiciliary service only as a temporary solution, and, although we cannot now create the real general-practitioner obstetrician immediately, that most valuable person can be evolved gradually upon the lines I have indicated. It is easy enough to encourage those who are interested in obstetrics, but it is extremely difficult to insist that the others should give up obstetrics altogether.

It has been said that "the price of progress is eternal trouble." If this is true then there is no cause for alarm; for if the amount of trouble that we are having over this particular problem is any guide to the future, then we should certainly make progress.

The Department of Health for Scotland has announced that a new formula for an improved protective midge cream has been worked out by the subcommittee on midge control of the Scottish Scientific Advisory Committee. This new cream, smeared on the exposed parts of the body, will ward off midges for at least three hours. It is more stable and more effective in giving protection against midge bites than the cream the committee introduced in 1946, and has the further advantage that it is not washed off by perspiration. The new formula is the outcome of investigation at the Royal Technical College, Glasgow, by Professor J. P. Todd and Mr. A. B. Gilmour, both of whom are members of the subcommittee. A large number of creams were prepared and tested on the human skin. The results showed that the new cream represented an advance on all others previously produced. Like the 1946 preparation, this new cream does not harm the skin, although there may be some slight tingling when it is first applied and afterwards when washing the face. It is recommended, however, that the cream should not be allowed to get into the eyes, nor should it come into contact with tortoiseshell or plastic spectacle frames. It has been decided to publish the new formula in the expectation that commercial concerns interested may undertake production without any proprietary restriction. If no manufacturing difficulties come in the way the publication of the formula may mean that the anti-midge cream can be on sale to the public this summer. The new formula is: dimethyl phthalate 200 g., magnesium stearate 30 g., zinc stearate 70 g. In preparation D.M.P. is first levigated with magnesium stearate, then zinc stearate is gradually incorporated.

WORLD FOOD PROBLEMS

LORD BOYD-ORR ON THE ABOLITION OF HUNGER

The Association of Scientific Workers arranged a largely attended conference at St. Pancras Town Hall on March 4 and 5 for a discussion on world food problems. Lord Boyd-Orr, president of the association, and other authorities on nutrition and agriculture took part. The delegates were from trade unions and councils, cultural societies, and two political parties (the Conservatives and the Communists).

Increasing Population

Dr. Julian Huxley, late director of Unesco, opened with a survey of the population problem. It had been the fashion in this country, he said, to speak of a possible decline in population, but that was only a local and temporary phenomenon, masking the real issue, which was the trend of world population as a whole. In all human history, apart from temporary periods of stagnation or of regression, the population had steadily increased, and not only so, but the rate of increase had risen. The present population of the world was 2,200 million; in 1800 it was 920 million; in 1650 about 545 million. Earlier computations suggested that at the beginning of civilization the population was 20 million, reaching 200 million by the middle period of the Roman Empire. The rate of increase was now 1% per annum; in earlier periods it was 0.1%. The net increase of world population was now roughly two persons in every three seconds, or something like 60,000 every twenty-four hours.

At least half, probably two-thirds, of the world's population were suffering from under-nutrition or malnutrition in greater or less degree. Even if the population remained stationary a great increase in food production would be needed to nourish it properly. The U.S. Department of Agriculture stated that in the ten years since 1939 the increase in population had been greater than the increase in food production. The population of a country seemed to follow a cycle: (1) a high birth rate approximately equalled by a high death rate; (2) high birth rate with lower death rate; (3) birth rate still higher than death rate, but both falling; (4) low birth rate equalled by low death rate; (5) birth rate lower than death rate. Asia and Africa were preponderantly in the first two phases; the rest of the world preponderantly in the last three. During the twentieth century Egypt had increased its population from 9 to 20 million. In Mexico the birth rate was still double the death rate, and both were high. The population of the U.S.S.R. was increasing by 2 million a year. He gave the following figures for the continents (in millions).

	1550	1940		1550	1940
Europe	100	540	Asia	330	1,150
North America ..	1	142	Africa	100	150
Latin America ..	12	131	Oceania	2	11

But while Europe and North and Latin America had expanded owing to new techniques and migration, Asia and Africa would in future have a much larger proportion of the world population.

What was needed, said Dr. Huxley, was a world population policy. Something like a change of heart was necessary, so that people would begin to think in terms of quality and not of numbers. At present the quantitative aspect was dominant in the minds of politicians and others. He hoped that before long a conference on population would be called by the United Nations.

Strategic Food Policy

Mr. F. Le Gros Clark pointed out that the increase in population was due not so much to the rise in the birth rate as to the rise in the survival rate, with the spread of knowledge of hygiene, sanitation, and health services, and particularly the control and eradication of killing diseases like smallpox, plague, and malaria. It was sometimes complained that medical discoveries and enterprise kept large numbers of people alive without ensuring for them the means of existence. There were two answers which could be made to such an accusation. In the first place it was just a waste of time to suggest that any brake could be put on these epidemiological discoveries and their

application. People in general knew that these diseases could be eradicated, and they were not going to refuse the help of medical science. The fact was that medical science was the pace-maker for agricultural science. But whereas malaria in regions like Cyprus or Sardinia or British Guiana could be cleared up in three or four years with a relatively small team and at an expenditure of half a million pounds, a commensurate advance in food production meant the enlistment of a vast army of peasantry all equipped for the purpose. The second answer was this, that the primary need in many countries was increased production per man, and if these killing diseases, including tuberculosis, were allowed to continue it would be impossible to get the working population on the land.

Mr. Clark held strongly that the problem was not to be solved by methods like birth control. To begin with, such methods offended the prejudices of vast numbers of people. To put them forward would mean a head-on collision with the Papacy and with some of the religions of the East. But, apart from religious and emotional reactions, it would be very difficult to instruct great masses of illiterate people in the effective use of contraceptive methods.

Of the population of the world about 1,500 million, or 70%, were peasants and cultivators in one form or another, and the large majority were oppressed by economic evils—lack of fertilizers and equipment, outmoded husbandry and land tenure, absentee landlords, unorganized markets, illiteracy, and lack of co-operation. Moreover, most of the good soils had already been used; those on which agriculture could expand in the future were marginal. The problem could be tackled only on a large co-operative level under the direction of state and local authorities. In many parts of the world it would mean not only a revolution in agricultural methods but a vast social change. He reminded the audience that a hundred years ago this country was inured to tales of recurring famine in India and China. Why this new sensitiveness? Was it because of the realization that this country was in a precarious state of balance, and that if outside supplies were cut off it could not hold out for as long as India? He pleaded for the building up by United Nations of strategic food reserves against famine.

Political and Economic Dangers

Lord Boyd-Orr, who was ill and whose paper was read in his absence, said that world production was back to about the pre-war level for grain and sugar, to within 5% of pre-war figures for fats and oils, and to within 8% for meat. But production and consumption had not moved in the same direction in all countries. In the United States the consumption of cheap foods like cereals and potatoes had fallen, and that of more expensive foods had risen. In European countries the consumption of cheap foods had increased, whereas the consumption of more expensive foods—with the exception of liquid milk in the United Kingdom—had declined. Before the war nearly two-thirds of the world population were not adequately fed. During the last ten years the population had increased by 200 million; consequently even if pre-war production were reached the amount available per head would be less than pre-war.

There were two dangers in the situation, one political and the other economic. The people of Asia, who had long borne poverty with resignation, had come to believe that if they took their countries into their own hands and established a new order they would abolish hunger and poverty. This upsurge of Asia was likely to spread to other continents. It was idle to say it was the spread of Communism. If Communism could deliver the goods no military force could stop its spread among ill-fed peoples. The economic danger was that with technical advances in food production unmarketable supplies of food were being accumulated in the United States, whose export market was being blocked by the inability of other countries to pay for them. The *Washington Post* had said the other day that the United States was already on the slippery slope of the 'thirties, with poverty in the midst of potential plenty.

These two problems, of scarcity of food bringing about revolt, and of unmarketable surpluses creating economic difficulties, should in any sane world cancel each other out. The specialist agencies of United Nations, such as the FAO, the World Bank, and the Economic and Social Council, were established to get nations to co-operate in producing a rapidly expanding world economy. In 1946 the FAO put forward proposals for a world food plan to double world production in the next twenty-five years.

"The doubling of world food production would call for an enormous volume of industrial projects, many schemes like the Tennessee Valley Authority, plans to stop soil erosion and recondition waste soils, tractors and farm implements, and vast quantities of consumer goods for the agricultural population.

"To carry out such a plan would require credits to countries to enable them to purchase the industrial equipment and to provide a world fund to ensure that everything could be sold. The World Bank was set up for that very purpose. If all nations were willing to co-operate in an agreed scheme of this kind for the development of the potential resources of the earth and to contribute to an international fund in proportion to their wealth, those having no money putting in an I.O.U. which would be honoured when their natural resources were developed, the world would be well on the way to the abolition of hunger, poverty, and preventable diseases and to agricultural and economic prosperity, with doubling and redoubling of world trade, and, most important of all, if the nations co-operated on a definite concrete plan of benefit for all, the ideological differences which separate nations would in time lose their significance."

Food Production in the Tropics

Dr. A. H. Bunting, who is chief scientific officer of the Overseas Food Corporation and formerly a member of the Nutrition Unit of the Medical Research Council, spoke on food production in the Tropics. No one really knew, he said, the extent of present world shortage, which varied from season to season. An estimate had been made by the FAO on the basis of the probable population in 1960, taking very modest nutritional targets—a level of calories which, if it were imposed in this country, would make people think there was something wrong with the Government. On these calculations the world production of cereals by 1960 as compared with 1939 must be raised by one-fifth, of roots and tubers by one-fourth, of sugar by one-eighth, of fats by one-third, of pulses by four-fifths, and of meat by nearly one-half. Twice as much milk would be needed, and two and a half times as much fruit and vegetables. This represented an immense quantity of food. It was true that technical improvements were possible in existing production, but optimism must be restrained by the realization that the majority of food producers, the world over, were on an extremely low level of production. Peasant farming, particularly in the Tropics, was not capable of much improvement unless accompanied by such great social changes that it would cease to be peasant cultivation as it was known to-day. He could not believe that by the year 2000, let alone 1960, this major transformation would come about.

New land must be brought into cultivation. Two main areas were possible—the extreme northern regions of the eastern and western hemispheres, and the tropical belt. It had been estimated that in the Tropics 600 million acres could be brought in. This figure was very questionable. Technical difficulties in the development of new land in the Tropics were apt to be underestimated. Nevertheless, there were good reasons for looking to the Tropics rather than to the other world reserves for future development. Big problems in Africa would have to be overcome—resistance to drought, the combating of pests, and the recruitment of many more scientists, such as entomologists and soil chemists. Attention must be paid to the social problem of resettlement, for unless in their new environment the people could maintain their productivity, the bush—and the tsetse fly—would come back.

Dr. Bunting marked out two great undeveloped regions of the world as offering potentialities for the future. One was the basin of the Amazon and the Orinoco in South America, which might be left to the technical resources of the Americans; the other was the still almost empty country of East and Central Africa. He contested the statement made earlier that all the fertile land of the world had been taken for agriculture and only marginal land remained. But in these tropical areas water was the first problem which would have to be tackled. He also considered that very large areas in East Africa were potentially

well suited for the production of rice, but here again the development of rice lands would call for extensive engineering work.

Agriculture at Home

Another important contribution to the symposium was made by Sir George Stapledon, who discussed ways in which by good husbandry and wise statesmanship the soil of Great Britain might be made to produce more food. Permanent grass, he thought, should not contribute more than 20% of the total of cultivable land. What could be achieved depended upon how many acres of permanent grass could with justice be turned into arable. A million acres of rough grazing land could easily be brought into regular arable cultivation, and another two million acres into what would be extremely good grass land.

Dr. G. A. Reay, superintendent of the Torry Research Station, Aberdeen, reminded the Conference of the vast resources of the sea as a source of food. The present annual consumption of fish was about half a pound (edible weight) for each person every week. Research was proceeding on expanded fish production in the arctic and other waters, but the seas were not inexhaustible, and consideration should be given to the best utilization of fish. In Great Britain with its network of railways the consumption of fish depended on rapid distribution of fish kept cool on ice or lightly smoked. Under the best conditions of storage white fish could be kept only for about a week, but with new freezing plant it could remain in good condition for a greatly extended period. Canning was also a method of long-term preservation, but it had its limitations and was not likely to take a key position in the utilization of fish in this country.

Mr. S. A. Barnett, of the Infestation Branch of the Ministry of Agriculture and Fisheries, gave some account of the work carried out for the effective protection of food from vermin. Finally, Mr. Morris Goldsmith described what Unesco is doing to encourage food production and conservation. At its conference in Mexico City in 1948 the director-general was instructed to request the member states, national commissions, and co-operating bodies to initiate in each country group discussions on the social and international implications of science, and the first topic proposed was food. This was now the discussion theme in fifty-two countries among their various associations for the advancement of science.

When the Conference, under Dr. D. McClean's chairmanship, was opened for general discussion there were many brief speeches, in which a diversity of views were put forward and many remedies proposed. Paul Rotha's documentary film, "The World is Rich," was shown as a prelude to the Conference.

Nova et Vetera

A PORTRAIT OF WILLIAM HUNTER

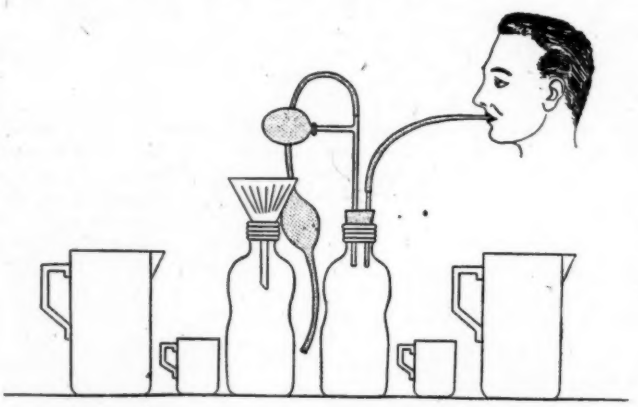
A portrait of William Hunter by Sir Joshua Reynolds has been acquired by the Hunterian Society. It is a portrait hitherto unknown and appears never to have been cleaned, so that the colours are not easily discerned. Experts, however, regard it as a portrait of high quality, and Mr. C. R. Rudolf, the curator of the Society's collection, said at a meeting of the society at the Mansion House on February 27 that it was one of the most important pictorial discoveries in the medical world for many years past. So far as is known there are only two other paintings of William Hunter by Reynolds in existence, both of them in the Hunterian Museum at Glasgow. They represent William Hunter lecturing on the gravid uterus, and are excellent pieces of pictorialism, but cannot be said confidently to convey a good likeness. The newly discovered picture is acclaimed as a fine portrait. On the back of the painting are written, in an early handwriting but not perhaps contemporary with the painting itself, the dates of the birth and death of William Hunter, and the statement that in the last year of his life (1783) the portrait was painted by Sir Joshua Reynolds. The picture is now being cleaned.

Preparations and Appliances

A SIMPLE GASTRIC SUCTION APPARATUS

Mr. M. D. SHEPPARD, F.R.C.S., surgeon, Chelmsford and Essex Hospital, writes: There are many ways of maintaining gastric drainage, but the only really effective method is the use of a powerful syringe applied to a wide-bore oesophageal tube at regular intervals. This way unfortunately is time-consuming and requires the constant attention of a member of the nursing staff. The various gadgets in use for maintaining constant suction are complicated and by no means foolproof, particularly when they have to be regulated by nurses who are not usually well versed in all the mysteries of hydrodynamics.

The simple apparatus illustrated here consists of a standard blood transfusion bottle in which a partial vacuum can be



maintained by using a Higginson syringe in reverse. Its valves are so placed that air is withdrawn from the bottle by about 40 compressions of the bulb, which is then allowed to hang down from its attachment to the vertical portion of the glass tube, thus kinking the rubber tubing and preventing a leak back through the valves. The single bent tube placed through the second hole in the rubber cork is connected to the stomach tube. Attached to the horizontal arm of the glass tube is a soft rubber bulb which remains collapsed while the bottle is sufficiently evacuated, its return to normal contour being an indication for further pumping by the nurse or the patient if he is co-operative enough.

On the same table can be kept another bottle of similar capacity into which is poured water equal in volume to any fluids taken by mouth, thus permitting a simple visual check on gastric intake and output. A fluid-balance chart should be fixed to a head-board near by.

This apparatus has given good service and can be rigged up quite conveniently with the usual equipment available in ward and theatre. The Higginson valves can be kept in working order by careful cleaning, by smoothing away any roughness on the seating or their bobbins, and by oiling them from time to time with a little liquid paraffin. Messrs. Allen and Hanburys have assembled the complete outfit and can supply it.

Regular employment is provided for severely disabled people in some special factories and workshops. Government departments help this scheme by placing contracts with organizations providing this sheltered employment. The Minister of Health has asked local authorities and hospital boards and committees to place contracts in the same way where possible, provided that goods of the quality and type required are offered at prices comparable with those offered by other suppliers on a reasonably competitive basis. A number of workshops for the blind are finding it difficult to keep the workers fully occupied, and it is hoped that they in particular may be helped in this way.

Correspondence

Maternity and the N.H.S.

SIR,—Professor James Young (February 18, p. 392) has ably stated the advantages of a maternity service in which there is "unity of control and unity of purpose," and he rightly expresses the fear that in the new service there may be "the very antithesis of the intimate and living collaboration which animates the ideal service." But, as he goes on to say, it is necessary to think "how far we can devise within the system . . . an adequate means of achieving a reintegration of purpose and effort." That surely is the crux of the matter, and just as surely the onus cannot be placed on the regional boards. They may devise a plan or bless a plan devised by others, but the problem must be tackled more intimately and locally.

In each group in the country there is probably already—and if not it is desirable that there should be—a key hospital which should be the focal point of the maternity services of the group. At this hospital there will be specialists, and to this hospital all practising obstetricians should have access.

In Group 5 (Woolwich) of the South-east Metropolitan Regional Hospital Board a scheme has been inaugurated whereby medical officers, midwives, and health visitors working in the county council clinics attend a monthly meeting at the hospital to discuss obstetric and administrative problems and to learn the fate of patients sent to the hospital and so on, and thus two portions of the service divided by a ministerial wedge are more firmly united than ever before. All the maternity hospitals in the group have a uniform system of booking which ensures that all who need the beds get them, and all who can have their babies at home do so, and there is easy transference of patients from domiciliary and hospital service and vice versa as required. In addition, the clinic medical officers in rotation come to live in hospital for a week-end and see what there is to be seen. Although this latter part of the scheme is in its infancy it promises well.

In time the practitioner will increasingly come into the orbit of the hospital. They do not wish to have to grapple with their problems alone and unaided. The way in which the practitioners avail themselves of domiciliary consultations is evidence of this. The dissemination of the Diplomas of Obstetrics will further this trend. We must expect single-minded theoreticians to produce peculiar plans, but with versatility at the receiving end there is no need for alarm.

I disclaim all credit for the scheme outlined. It was started, I believe, in the Greenwich and Deptford Group with Mr. Alistair Gunn's support, and has also been started in the Bromley Group. Believing it to be necessary and good, all I have done is to welcome it in the Woolwich Group, and I strongly recommend it to any who have not already got a similar scheme in operation.

From the inception of the National Health Service Act Sir Allen Daley has been most keen in instituting and cementing association with obstetric units, and the credit must go to him for his proposals and to his divisional medical officers for their implementation on the lines described above.—I am, etc.,

London, S.E.3. KEITH VARTAN.

SIR,—Professor James Young's inaugural lecture at the Royal College of Midwives in October last has made a delayed but welcome appearance in your pages (February 18, p. 392). It is an admirable description of the dismemberment of the maternity service which had been developing over many years on the principle of providing "unbroken continuity and singleness of control."

The success of that principle found its most striking vindication for some of us in certain large emergency maternity schemes during the war. Team-work of the high order then put into practice would be impossible now, but it was towards such a standard that normal maternity schemes were progressing. Professor Young is far from exaggerating the present position. In one county at least there are five authorities involved, and nowhere does the present system—if such it can be called—give promise of simplicity for the future.

Unless the ground gained by long and patient endeavour is to be lost there must be a reintegration of the service on the lines suggested by Professor Young. That object is surely one for the Royal College of Obstetricians and Gynaecologists supported by all who realize the inevitable effect of existing conditions.—I am, etc.,

St. Mawes, Cornwall. CLAUDE TANGYE.

SIR,—Many general practitioners will disagree with some of Professor James Young's remarks (February 18, p. 392).

1. "It has long been recognized that for normal delivery the general practitioner with his many other commitments is less suited." By whom has it "long been recognized," may one ask? Certainly not by the patient. Many thousands of the 72% were delivered by midwives because they could not afford a doctor, and for no other reason. And why is he "less suited," pray, when for years he has been at hand for the midwife to fall back on when she is in a difficulty?

2. If the midwife is to be "degraded to the status of the maternity nurse"—and I think the phrasing most unfortunate—there again it is the patient's wish. When one is booked for a confinement under the N.H.S. one is expected to do a little more than an antenatal or two and a post-natal at the sixth week for one's seven guineas. Very many patients of the middle- and upper-income groups who had their babies as private patients before 1948 are now having them under the N.H.S. They demand, and rightly so, the same personal attention as before.

3. Who are we as a profession to advocate "midwives only" for normal domiciliary cases, when in our wives' confinements we should not dream of not having an able and experienced colleague in attendance?—I am, etc.,

Aylesbury, Bucks. W. A. BELLAMY.

SIR,—Professor James Young's article on "Maternity and the National Health Service" (*Journal*, February 18, p. 392) prompts one to take up the whole question of the G.P.-specialist relationship.

On all sides one hears the constant lament from one's G.P. colleagues that one is becoming no more than a glorified or unglorified sorting clerk. It would appear that the profession as a whole regrets this, but the tendency continues. The G.P. has no professional association of G.P.s as such, and is quite unable to arise and defend himself.

Each specialist decides that on no account must the G.P. partake in his specialty. To quote two examples only: the anaesthetist has decided that anaesthetics are the exclusive province of the specialist anaesthetist; the G.P., who for years gave anaesthetics quite successfully, is to be pushed right out. A second and more surprising example occurred to us when attempting to make an appointment for a child at the local hospital. We were informed that the child could be sent to a special department only by the "Child Health." Apparently the G.P.'s diagnostic skill ends at being able to say whether a child is above or below 12.

Now Professor Young and obstetricians in general make one wonder whether it is really interest in the midwives' status that they are concerned with, or is it too presumptuous to think that there might be a little fear of competition from the G.P.-obstetrician? If it is the fall in status that is to be lamented, then surely it is the fall in status of the G.P. Bad midwifery caused by the pressure of work can hardly be blamed on the bulk of careful G.P.-obstetricians. If it does occur, then who would suggest that it occurs any less often with the obstetric specialist?

If the G.P. is to be portrayed as an ogre who applies forceps without any consideration of judgment, then would the obstetrician specialist claim that he cannot be accused of many hundreds, even thousands, of unnecessary caesarean sections, all, remember, carrying, even if small, some maternal mortality. If meddlesome midwifery is to be shrieked at the G.P., then perhaps it would be to the point to ask whether all caesarean sections, inductions, etc., performed by the specialist were necessary.

This is the time for the G.P. to speak to his specialist colleague as a colleague and as an equal. Surely the field of midwifery is big enough to give scope to all who are interested. If there is any point in the specialist obstetrician's attending the perfectly normal confinement, the only reason for his attendance being the private fee, then surely it is proper for the G.P. to attend the patient he has known and is respected by, and by whom he is looked to as a counsellor and friend. If the woman in labour is given solace by his presence, surely that in itself is very much in his favour. The sneer that the woman feels safer when her doctor is present can hardly be taken seriously. We would imagine that the presence of the practitioner capable of dealing with an emergency, if it arises, is something to be desired. Or are we now coming to the stage that, when Lady So-and-So, capable of paying a very fat private fee, applies to Mr. So-and-So, he will inform her, "You will be quite safe with the district midwife"? No, Sir, the G.P. has a very definite place in midwifery, and it is for him and any professional association that he belongs to to fight to maintain that place.

If Professor Young is really interested in continuity of services, then surely he could not have failed to see that the simplest answer is that the G.P. shall be entitled to demand, as of right, an afternoon at the local antenatal clinic; that he shall continue to do midwives' emergencies as hitherto; and, to make the link-up complete, he should be allowed a case or two under his own care at the local hospital. Who can suggest that this would not be a much more practical and satisfactory arrangement? Continuity would thus be complete between midwives, doctor, resident, and visiting staff. It seems farcical to ask the wholly inexperienced clinic M.O. to do obstetric emergencies, and at the same time decry the G.P. with many years of practical obstetric experience. The field is surely big enough for all, and who will benefit from the process of ousting one's neighbour?—We are, etc.,

M. CHALK.
E. JOZEF.

Gateshead.

The Psychology of the Surgical Patient

SIR,—I should like to congratulate Professor Alexander Kennedy on his article, "The Psychology of the Surgical Patient" (February 18, p. 396). As he says, there is nothing very new, but the article is full of matter which we are liable to forget and of which it is good to be reminded.

In particular I wish to draw attention to the section "The Patient's Point of View," which all surgeons recognize but to which few have time to attend. My object in writing this letter is to suggest a second-best method of dealing with this problem. Would it not be possible for every patient on entering a hospital to be given a short pamphlet which would show that the staff realizes the patient's point of view, and would give a warning of those little difficulties that are likely to occur in a new set of surroundings? I can think of no one better qualified to compose such a pamphlet than Professor Alexander Kennedy. Perhaps the King Edward's Hospital Fund for London would publish it.—I am, etc.,

MALCOLM DONALDSON.

London, W.1.

Ogilvie's Syndrome

SIR,—Further to the articles by Mr. J. A. MacFarlane and Mr. S. K. Kay (December 3, 1949, p. 1267) and Sir Heneage Ogilvie (October 9, 1948, p. 671) on the subject of spastic ileus or false colonic obstruction, I should like to add a recent case of this occurrence to the literature.

A man aged 73 was admitted to my ward on November 16, 1949, with the following history. Five days previously he began to complain of severe colicky pains across the lower abdomen. Constipation was complete, associated with nausea, but no vomiting, and the fullness of the abdomen had noticeably increased. He had not experienced any previous attacks.

On examination, his tongue was furred but moist. The abdomen was tense and greatly distended, with tenderness in the right lower quadrant. Borborygmi were clearly heard and rectal examination was negative. The diagnosis of acute large-bowel obstruction probably due to carcinoma was made and immediate laparotomy performed. The abdomen was opened through a right lower paramedian incision. The caecum, ascending, and transverse colon were greatly distended as far as the splenic flexure, whence the dilatation subsided. A careful search did not reveal any organic cause for the obstruction, and I remarked to my assistant that I hoped a lesion of the splenic flexure was not being missed. A similar thought was expressed by MacFarlane in his first case. The distension was so pronounced that a transverse colostomy was performed through a left upper rectus incision.

The patient's condition deteriorated, and he died four days after operation. The post-mortem examination did not reveal any organic lesion of the bowel which may have caused the colonic obstruction, nor was the coeliac plexus involved in any malignant process.

The spastic obstruction occurred at exactly the same site as in MacFarlane and Kay's first two cases—i.e., at the splenic flexure. Is this the site of maximum neuromuscular imbalance, where perhaps the vagi and middle colic sympathetic plexus meet the corresponding sacral autonomic outflow and the left colic sympathetic nerves? Although the cause of the obstruction is not mechanical, one does not know how long the spastic ring will persist, so a colostomy will be advisable in most cases.—I am, etc.,

ALFRED M. ABRAHAMS.

Liverpool.

Experiences with "Diparcol"

SIR,—Dr. R. M. Gray, in his medical memorandum about the treatment of Parkinson's syndrome with "diparcol" (February 18, p. 416), refers to various methods of control—"dummy tablets containing lactose and also capsules of 'benadryl' being substituted." In my own limited experience (one case) the use of benadryl (250 mg. daily) alone was equal in effect to diparcol (1.25 g. daily) alone. Maximum benefit was obtained using a combination of benadryl 150 mg., diparcol 0.75 g., and amphetamine 15 mg. daily. In the circumstances it does not seem justifiable to use benadryl as a control if the aim is to estimate whether or not any psychological improvement has followed the administration of a capsule regardless of its chemical nature.—I am, etc.,

F. MURRAY.

Uffculme, Devon.

Red Blood Cell Diameters

SIR,—Drs. W. H. H. Merivale and G. O. Richardson (February 25, p. 463) write that they measured the mean corpuscular diameter "by the method of Price-Jones (1933), dry films stained with Leishman's stain being used." It may be unimportant, but Price-Jones in the quoted reference says that he always fixed and stained his films in Jenner stain for two minutes, and, after washing and drying, superstained with a weak aqueous solution of eosin for a further two minutes. He adds the comment: "These details should always be adhered to, since it is found that alterations in the fixing and staining reagents can produce changes in the mean diameter of the cells." In his laboratory there was no crime more heinous than the use of "my eosin" for any other purpose.—I am, etc.,

J. A. BOYCOTT.

Taunton, Somerset.

Antibiotics and Hydrogen Peroxide

SIR,—During the last five years I have treated about a dozen cases of Vincent's stomatitis by applying a combination of either buffered lactic acid or calcium lactate on one side, and H_2O_2 or sodium percarbonate or perborate on the other side. The results achieved by this method compare favourably with those produced by penicillin, especially so far as speed of action is concerned.

The form of application was simple. The patient rinsed his mouth four to six times a day with a solution consisting of a teaspoonful of calcium lactate or of buffered lactic acid in a beaker of water to which a teaspoonful of H_2O_2 (20 vol.) or, when using the lactate, half a teaspoonful of the per-salt was added. The solution was freshly prepared each time. This method of treatment might prove useful in cases of Vincent's infection refractory to penicillin treatment. I am publishing it, however, not so much for this reason as for its possible theoretical implications.

Lactates, as such, have no effect whatsoever on Vincent's infection. The effect of H_2O_2 on the disease is known to be at best very limited. The results achieved by a combination of both, however, are so striking, usually noticeable within 24 hours, that the idea of some specific action suggests itself. I have formed the hypothesis that the lactic acid acts as an H-donator, and that the hydrogen produced from it by lactic dehydrogenase, together with the oxygen from the H_2O_2, forms H_2O_2 again.

This hypothesis seems to be supported by the finding of Blanchard and his colleagues[1] that H_2O_2 is formed when lactic acid is oxidized catalytically in the presence of molecular oxygen. Saliva is known to contain catalase; presence of lactic dehydrogenase in saliva has been shown by Eggers-Lura.[2] Though, as mentioned above, H_2O_2 is of little value as an antiseptic when introduced from without, it has been found by various authors in recent years that some micro-organisms are extremely sensitive to minute amounts of it if it is formed in the culture medium or in the tissue itself. The catalase possessed by these micro-organisms is unable to protect them against the H_2O_2 thus formed.[3-7]

It has further been shown by various authors that preparations which are capable of forming H_2O_2 by enzymatic action have an antibacterial effect. Lipman and Owen[6] found that the system hypoxanthine–xanthine oxidase inhibits bacterial growth owing to formation of H_2O_2. Lwoff and Morel[7] and Keilin and Hartree[3] ascribe the antibacterial action of ascorbic acid to the H_2O_2 formed by the oxidation of this compound.[8 9] Green and Pauli[10] have pointed out that any flavoprotein enzyme system that produces H_2O_2 shows antibacterial activity. At least one antibiotic, notatin or penicillin B, has been shown to owe its antibacterial effect to its producing H_2O_2. Notatin in dilutions as high as one part in one billion inhibits the growth of *Staph. aureus*.[5]

In view of all this it does not seem unreasonable to suggest that it might be possible to produce synthetically a preparation with properties similar to those of an antibiotic by making a compound containing a group capable of acting as an H-donator—e.g., a lactic or succinic group or other group with a mobile H-atom such as SH, NH_2, NH, or the like—and containing at the same time a group capable of supplying oxygen as an H-acceptor. Also, the mode of action of one or the other of the natural antibiotics might be explainable in a similar way.—I am, etc.,

Twickenham, Middlesex. A. KRAUS.

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Tetanus Treated by "Myanesin"

SIR,—Cases of tetanus have been reported which illustrate the beneficial results of tubocurarine[1] and "myanesin"[2] given intravenously and intramuscularly. We recently saw the following case, in which, owing to paucity of veins and their small size, it was clear that continued intravenous therapy would be well-nigh impracticable.

A boy aged 6 years 10 months injured his left big toe seven days before admission. Sore throat and abdominal pain developed the night before admission. Trismus and rigidity of the back were noted the following morning. Rigidity increased and opisthotonos developed: spasms also occurred the same evening. Treatment along the usual lines with rectal paraldehyde, penicillin, antitetanic serum, and toilet of the wound was carried out. After 48 hours the child was still in a critical condition with marked rigidity and numerous spasms despite sedation. There was difficulty in swallowing, and rectal salines had to be given. Myanesin elixir was then started: rectal administration was first used, since the elixir irritated the patient's sore mouth—the result of biting his tongue. The dose given was 2 dr. (7 ml.) per rectum four-hourly, increased to 4 dr. The oral dose, given when the condition permitted, was 2 dr. Treatment was continued for four weeks until the spasm had cleared.

Intramuscular and intravenous therapy both require frequent administration and entail painful stimuli which it is desirable to avoid. In this case it was decided to employ a preparation which could be given orally or per rectum, and myanesin elixir was used. Since the patient bit his tongue in the course of numerous spasms, oral administration proved too irritant and did, in fact, induce spasms. The preparation was effective per rectum and was easily administered by this route, either alone or in conjunction with rectal paraldehyde—four-hourly administration was adequate.

Our observations coincide with those of Belfrage,[2] that the severity of spasms was considerably diminished though their frequency was unaltered. The degree of general rigidity was reduced, particularly the trismus, and accordingly an early return to oral feeding was made possible. Inadvertently, a dose of myanesin elixir was omitted, and the sister, who did not know of this, reported a marked increase in rigidity. A minor troublesome side-effect was that voluntary micturition was often not possible when the patient was under the influence of myanesin. This could be overcome by lengthening the interval between doses to eight hours. Treatment certainly reduced the exhausting effect of the muscular rigidity and the spasms. This was a severe case with an incubation period of seven days and a period of onset of less than twelve hours, and we consider recovery would not have occurred without this treatment.—We are, etc.,

P. P. FOX.
Yeovil, Somerset. A. H. BANTON.

REFERENCES

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2 Belfrage, D. H., *ibid.*, 1947, **2**, 889.

Q Fever in England

SIR,—In your leading article (February 25, p. 473) on Q fever in England you mention that Stoker[1] found immune bodies to Q fever in "two R.A.F. men who had never been abroad." This statement is not quite accurate, inasmuch as one of the patients had been a prisoner of war in Germany. However, this case was recovering from an illness identical with Q fever at the time that the immune bodies were found in his blood. The second case had been in the same camp as the first case, had never left England, and had recently recovered from "atypical pneumonia." Unfortunately, he had been treated at a different R.A.F. hospital from the first, and it was only by the co-operation of the Director of Hygiene, R.A.F., that he was discovered. The full clinical details of these cases have been published.[2]—I am, etc.,

London, W.5. M. D. WARREN,
Squadron Leader, R.A.F.

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The Acute Throat

SIR,—It may be of interest to record that in contrast to Captain E. L. W. L. Lombay (January 28, p. 250) my experience as medical officer in charge of a tonsillitis ward led me to use sulphonamides as routine treatment for the acute throat in preference to penicillin. The correspondence in your columns which followed the omission of sulphonamides in favour of penicillin in the treatment of tonsillitis described by Dr. T. Anderson (October 15, 1949, p. 860) was reassuring. No one doubts the efficacy of penicillin in treating the acute throat, but as most cases of tonsillitis are treated at home, where frequent visits for penicillin injections are not possible, any limitation of sulphonamide therapy should be carefully studied. Even so I consider the following general observations made during an epidemic in Catterick Camp a year ago worth noting.

With normal dosage of sulphonamides the temperature in a typical acute throat falls sharply and is normal after two days. After three days the tonsils are usually clean, the tonsillar glands are frequently no longer tender, and the drug can be discontinued. Frequently the patient is back on full duty a week after starting treatment. This compares favourably with twice-daily injections of penicillin, and with Dr. Anderson's

statement that penicillin "must be continued for seven days to secure the best results." Further points in favour of sulphonamides are (1) that given the choice most patients prefer oral therapy to injections, and (2) the cost of sulphonamide therapy is less than half that of using penicillin.—I am, etc.,

Barnard Castle, Co. Durham. E. J. FAIRLIE, Capt., R.A.M.C.

Erysipeloid

SIR,—I have noticed recently the correspondence about erysipeloid infections of the hand and would like to add my observations of those cases seen in the last twelve months in North Queensland. Each case followed a puncture wound of the sole of the foot with a meat bone. The typical advancing reddish-purple edge proceeded in a fan-like manner over the dorsum and medial arch.

The course in two cases was three weeks. The other is still under treatment, but the condition has already been present for one week and is still spreading. I have noticed the absolute insensitivity to sulphonamides and feeble response to penicillin given as an oily injection of 250,000 units daily. The disease seems to cause little discomfort, but in the early stages there has always been a history of enlarged regional lymph nodes.

For a tropical region where fungus and other infections are alleged to abound one sees comparatively little of this condition.—I am, etc.,

Townsville, Queensland. C. J. LUKE.

The Wrong Solution

SIR,—Your medico-legal report entitled "The Wrong Solution" (February 25, p. 496) on the fatal error of using solutions of 1/1,000 adrenaline in the place of the *B.P.* strong injection of procaine and adrenaline points a lesson not only to hospital authorities.

Some years ago I was preparing in a hospital to suture a lacerated perineum under local analgesia. The nurse held out a bottle of adrenaline solution, but the bottle looked so like those of the procaine preparation I had been using for many months that I filled my syringe without checking the contents. Fortunately I did then read the label before beginning the injection. The point was that both types of solution were kept in bottles of identical shape and size and with printed labels of the same pattern and superficial appearance issued by one well-known firm.

Subsequently, in practice, I have been obtaining these preparations from another big firm, and here again the bottles and coloured labels appear just the same until they are very closely read. I now take care to stick a large red warning label on my bottles of 1/1,000 adrenaline solution. I should like to suggest that the issuing firms suitably alter their labels and also that until this is done practitioners suitably mark their bottles.—I am, etc.,

Cambridge. A. S. PLAYFAIR.

Aerosol Treatment with Penicillin

SIR,—I am very interested in the results of Mr. A. E. Bernstien (February 18, p. 441), who records outstandingly successful results from the aerosol treatment with penicillin in a series of 100 cases of bronchitis and asthma. I think that in a case like this the subject should not be discussed so briefly, and I plead with Mr. Bernstien to publish his results in full detail if his time permits, as by so doing I think he would be doing an undoubted service to his colleagues. In particular, I would like to ask him the following questions.

1. Was the treatment given individually or by group methods (i.e., by the individual face-mask or by penicillin infusion into a closed room seating several patients together)?
2. What were the ages of the patients treated and what was the severity of the asthma?
3. What was the time of one exposure to the vapour?
4. How did the follow-up of the cases compare with the apparently brilliant cures noted after the five treatments of 500,000 units of penicillin aerosol?
5. Were the cases treated in a hospital or at home?
6. Was any other treatment used simultaneously?
7. Were any control cases used?
8. Were any assays of penicillin in the plasma made, and, if so, were bacteriostatic levels found (i.e., over 0.07 unit per ml.)?

I once attempted mass penicillin aerosol therapy in a group of bronchitic and asthmatic children attending an open-air school, and although I infused 4 mega units into a small room of 900 cu. ft. (25.5 cu. m.), the children remaining in the room at the full concentration of 4,000 units per cubic foot for one hour, no penicillin levels in the blood were ever obtained, and I reluctantly gave up the method.

I wonder if Mr. Bernstien has used the mass method in any of his 100 cases treated. The literature is fairly plentiful on aerosol penicillin therapy in individual cases and in the oxygen tent, but is scanty, so far as I am aware, on group treatment such as I attempted to carry out. I should be very grateful for any information on this particular method, as it seems to me that there is a case for it, if effective blood penicillin levels can be reached, in open-air schools and other institutions where chronic chest cases are assembled for long periods.—I am, etc.,

Rotherham. E. H. JEANES.

Fatal Air Embolism in Artificial Pneumoperitoneum

SIR,—I have read with great interest the account by Dr. E. G. Sita-Lumsden and Dr. R. H. Elphinstone (January 21, p. 166) of the case of a fatal embolism in artificial pneumoperitoneum. They choose as their site of election for the induction of pneumoperitoneum, and for refills, a position below the left costal margin at the lateral border of the rectus sheath. I have performed the procedure many times and have seen it done often, and it has always been performed in the right iliac fossa at the outer border of the rectus sheath usually one or two inches (2.5 to 5 cm.) below the umbilicus. By this route the inferior epigastric vessels are avoided. The intestines, as is well known, recede before the advancing needle. In this route there are no large important structures encountered—i.e., liver or spleen, falciform ligament, or gastro-hepatic omentum.

I have never seen or heard of any fatal accidents when the above procedure was performed. Intraperitoneal adhesions do not prevent the circulation of air under the leaves of the diaphragm, and satisfactory results—i.e., air under the right diaphragm and pushing up of the diaphragm—have always been obtained. As far as I know the procedure I have described is a common one in use.—I am, etc.,

Jamaica, B.W.I. V. G. RADCLYFFE

Anaesthesia for Fenestration Operation

SIR,—I was very interested in Dr. Brenda G. Hutchinson's letter (February 18, p. 436) on anaesthesia for the fenestration operation. I agree entirely that adequate spraying of the pharynx and cords is essential, and that an endotracheal tube should be used to avoid any obstruction and consequent congestion. As to anaesthetic technique, however, I feel that the "semi-closed" technique described is not the one which produces optimum conditions. In my experience an "open" system reduces resistance to breathing and results in still quieter respirations with no congestive bleeding.

Having attached a Rowbotham connexion-piece to the endotracheal tube, a Schimmelbusch mask (covered with one layer of lint) is applied to the face and retained in position by zinc-oxide plaster. Ether–oxygen is insufflated under this mask by a long rubber tube leading direct from the Boyle's machine. This is similar to the technique which is used by Tumarkin, of Liverpool, and it gives results as pleasing to the surgeon as they are to the anaesthetist.

As to the use of adrenaline with trichlorethylene, the work of Barnes and Ives[1] would suggest that this is, to say the least, unwise, and I feel equally light anaesthesia can be maintained using ether and an "open" technique, and with greater safety to the patient. Finally, I would like to reiterate that satisfactory anaesthesia can enormously improve the surgeon's operating conditions in the fenestration operation.—I am, etc.,

Manchester, 20. M. SWERDLOW.

REFERENCE

[1] *Proc. R. Soc. Med.* 1944, **37**, 528.

SIR,—Dr. Brenda G. Hutchinson's letter (February 18, p. 436) on anaesthesia for the fenestration operation for otosclerosis prompts me to put on record the methods we have used at King's College Hospital for Mr. T. Cawthorne's cases. The following factors have influenced the choice of anaesthetic.

1. Bleeding must be at a minimum, for excess bleeding reduces the speed of the operator, adds trauma by necessitating the constant use of the sucker, and jeopardizes the success of the operation if it occurs during the making of the fenestra, for blood entering the fenestra aggravates post-operative labyrinthitis and may therefore reduce the decibel gain in hearing.

2. Only a light plane of anaesthesia is required, but there must be complete immobility of the head when pain stimulus is increased—i.e., during incision of the skin, separating the periosteum from the mastoid bone, inserting the retractor, curetting close to or in the cavity of the middle ear, and cutting the tissue flap from the wall of the auditory canal.

3. Diathermy and other electrical equipment are in constant use, hence a non-inflammable agent must be used.

4. Labyrinthine disturbance of some degree is inevitable, and therefore the anaesthetic agent must not initiate or aggravate vomiting.

Local analgesic as the sole agent was decided against from the outset, and its use has been confined to a preliminary infiltration of the skin incision with 1% procaine and 1 in 90,000 adrenaline. The operation is a delicate one and requires skill and concentration on the part of the surgeon, and the strain of a conscious patient may prevent the exhibition of either or both of these qualities: further, to have to supplement local analgesia with some form of general anaesthetic half-way through the operation (this happens in 25% of cases in America when local analgesia is used) is unsatisfactory, to say the least. Again, very heavy premedication is necessary to prevent psychological trauma when using a local analgesic technique—so heavy that one wonders if the local analgesic is really required at all. The use of black coffee and local analgesia as recorded by Dr. Hutchinson is interesting: the route of administration of the coffee, however, is not mentioned.

The method used from April, 1947, to March, 1948, was as follows:

Premedication of "omnopon" gr. ½ (22 mg.) and scopolamine gr. 1/150 (0.4 mg.) was given one hour before the operation, and at the same time a "decicain" lozenge was given to the patient to dissolve slowly in the mouth. Unconsciousness was induced with 5% thiopentone, using 0.5–1 g. according to the effect on the patient, and intubation was carried out under direct vision with a Macintosh laryngoscope and a No. 9 or No. 10 Magill's oral tube. The oral route is preferred to the nasal one, as the size of the tube is not thereby limited by the size of the nose. Also 10% "nupercaine" ointment was used on the intratracheal tube—1% is only a good lubricant. Anaesthesia was then continued with nitrous oxide, oxygen, and minimal trichlorethylene, and I endorse Dr. Hutchinson's emphasis on the small amount of "trilene" required.

This method, used in 118 cases, proved satisfactory from most points of view, but troublesome bleeding occurred in about 33% of the patients. In order to prevent straining on the intratracheal tube the anaesthetic had to be kept moderately deep, and patients were being very sick in the immediate post-operative period.

Hershey and Jones[1] in America advocated the use of "avertin" as a basal narcotic in order to lower the blood pressure, whilst Harris and Hale[2] even went to the length of bleeding their patients from the radial artery until the blood pressure was down to 80 mm. Hg and retransfusing the blood at the end of the operation, so anxious were they to control bleeding during the fenestration operation. We were not convinced that high blood pressures were the sole cause of excess bleeding, but felt rather that inhalation anaesthetics and the presence of an intratracheal tube were most often the cause. Hence it was decided to change our method to one of intermittent thiopentone and to dispense with the intratracheal tube. It was also felt that the prolonged sleep following thiopentone anaesthesia would be an advantage to a patient with labyrinthine disturbance and probably diminish the incidence of immediate post-operative vomiting. Since March, 1948, we have anaesthetized 229 cases by this method for the operation of fenestration and the allied operations of labyrinthectomy and facial nerve decompression. The following are the details of the technique:

Premedication was exactly as for the former technique, but in addition the nose was sprayed with 10% cocaine. Unconsciousness was induced with 5% thiopentone, using 0.5–1 g. according to the response of the patient, and the airway was maintained with bilateral nasopharyngeal tubes (Magill's nasal intratracheal tubes cut to the required length) and an oropharyngeal airway. Anaesthesia was continued with 2½% thiopentone given into an ankle vein—a 20 ml. syringe being strapped in position. The indication for adding more thiopentone is the movement-response test—i.e., the withdrawal reflex of the foot, which always occurs before any movement of the head (these reflex movements of the foot can be anticipated knowing the stage of operation reached). The average amount of thiopentone used for these 229 cases was 1.88 g.

There are few contraindications to this method, as most patients for the fenestration operation are fit adults between the ages of 20 and 45. Varicose veins, asthma, chronic nephritis, and pregnancy have prevented the method from being used in six cases. The results of the method have been very encouraging. Troublesome bleeding occurs very occasionally (about 10% cases); no difficulty with the airway has been experienced; the plane of anaesthesia can be immediately adapted to the requirements of the stages of the operation; and the incidence of immediate post-operative vomiting is almost *nil*. The purist might object to the method on the grounds that intravenous anaesthesia should not be used when the anaesthetist has not full control of the head. I feel, however, that the special problems of this very specialized operation have justified the development of this anaesthetic technique with benefits to both surgeon and patient.

My thanks are due to Dr. Vernon F. Hall, with whom this technique was developed and who administered half the anaesthetics in the cases reviewed, and to Mr. Terence Cawthorne for help and advice at all times.

—I am, etc.,

London, S.E.5. STANLEY A. MASON.

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[1] *Cleve and Clin. Quart.*, 1947, **14**, 159.
[2] *Arch. Otolaryngol.*, 1947, **46**, 390.

SIR,—I cannot claim experience of anaesthetizing cases for the fenestration operation which is comparable with that of Dr. Brenda G. Hutchinson (February 18, p. 436), but during the past four years I have been anaesthetizing cases for fenestration for Mr. G. A. Fraser. At first I employed a technique similar to that of Dr. Hutchinson, except that I always performed oral intubation in order that the largest possible size of Magill tube might be used.

Since discussing the problem of providing a blood-free field with Dr. G. P. Fox, who has great experience in anaesthetizing for this operation, I have employed a technique which he suggested to me. The average healthy adult receives a total of 6 g. of "nembutal," together with "omnopon" gr. ½ (22 mg.) and scopolamine gr. 1/150 (0.4 mg.) before operation. The patient is not intubated, but a nasopharyngeal tube is used, and anaesthesia is maintained with thiopentone given intermittently through a Gordh's needle. A total of 1½ to 2½ g. of thiopentone is used in the course of the operation, the blood pressure is recorded regularly and remains in the region of 90 mm. Hg systolic and 60 mm. Hg diastolic. Since employing this technique the bleeding from the operation site has been noticeably less than when gas–oxygen and trichlorethylene was used. Recovery is certainly not rapid, but there have been no complications that could be attributed to the anaesthetic used.—I am, etc.,

Hove, Sussex. REX BINNING.

Insufficient Anaesthesia

SIR,—The power of voluntary movement disappears under anaesthesia before consciousness is lost, quite apart from the use of curare. I well remember being given an anaesthetic for a minor operation more than thirty years ago. The fashionable induction at that time was a chloroform and ether mixture, with the latter predominating. I was still fully conscious, but could not move or speak, when the surgeon asked: "Isn't she

ready?" My feeling of gratitude towards the anaesthetist can be imagined when he replied, "Not quite." I knew that he had lifted my eyelid, as I recognized the difference between light and darkness, although vision had gone. Very soon afterwards I was in a deep sleep which lasted until I awakened in bed. But it was an unpleasant moment, and it made me much more careful subsequently in my choice of conversation during the induction of an anaesthetic.—I am, etc.,

London, W.8. VIOLET RUSSELL.

SIR,—Dr. E. H. Winterbottom's report upon his case (January 28, p. 247), in conjunction with recent case reports by other writers upon the use of curare as an adjunct to anaesthesia, seems to me to point the need for clarification and understanding upon at least three aspects of this anaesthetic technique.

The first naturally relates to the optimum plane of anaesthesia to be employed in conjunction with curare. This will naturally vary with the severity and duration of the operation, but it is my contention that first-plane anaesthesia, that most often cited and advocated, is insufficient to guard against reflex effects and shock where the operation is a major proceeding. I believe that nothing short of upper second-plane anaesthesia should be maintained.

The second relates to the more difficult problem about the best anaesthetic agent to employ in conjunction with curare. Nitrous oxide with oxygen alone is definitely unsuitable. In conjunction with thiopentone two difficulties arise: first, it is impossible to be certain what depth of anaesthesia is being maintained, and, secondly, the plane of anaesthesia is unstable unless the thiopentone is administered by constant drip. The great advantage that thiopentone has over the volatile agents is that the depth of anaesthesia is unrelated to the tidal volume and therefore remains constant in spite of assisted or controlled breathing. With ether, "vinesthene," and cyclopropane the difficulty again arises in accurately assessing the depth of anaesthesia in the curarized subject, and, in contradistinction to thiopentone, once assisted or controlled breathing becomes necessary, as one or other always does when curare is employed, all accurate control over the depth of anaesthesia is lost.

Of the volatile anaesthetic agents, I am convinced that trichlorethylene, in conjunction with nitrous oxide 70% and oxygen 30%, is the best agent to employ in the majority of cases. By virtue of trichlorethylene's low volatility it is extremely easy to maintain an even plane of light anaesthesia which is relatively unaffected by assisted or controlled respiration. It is difficult to exceed second-plane anaesthesia with trichlorethylene, and in the first plane the high analgesic quality of this agent is present to protect from undue shock. Moreover, it is my experience that unpleasant anaesthetic sequelae are extremely rare after the use of trichlorethylene.

My third point relates to the very high doses of curare which it seems are commonly employed. My experience in the use of curare, which dates from the time when the original crystalline product was first available, has led me to regard an initial dose of 12 to 15 mg. followed by additional doses of 2.5 mg. at intervals of twenty minutes, lengthening to periods of thirty or more minutes in prolonged operations, as sufficient to maintain full relaxation for any purpose in the average robust adult. Upon this basis such doses as 65 mg. are beyond my comprehension, and I find it hard to credit the statement that the respiration of a patient to whom such a dose was administered over 4½ hours was "at no time less than 20% of the normal excursion." Reference to my records shows that the maximum dose I have employed is 30 mg. in 5½ hours and that with this dosage controlled respiration was necessary at times.—I am, etc.,

Bournemouth. S. F. DURRANS.

SIR,—I feel it is time I intervened in the correspondence about curare in anaesthesia started by Dr. Eric Winterbottom (January 28, p. 247). Dr. Winterbottom showed me the letter before he submitted it for publication, and it seemed to me that it might be helpful to put on record the event.

The points I feel called upon to clarify are the following. First, that at the time the patient became aware of what was going on she gave evidence of being, from the surgeon's point of view, incompletely anaesthetized, and I asked that she should be put a little deeper. Secondly, the memory about which the patient spoke subsequently was of severe pain, but she spoke of it as a matter of interest rather than of concern. It would be the greatest error to leave the impression that she had sustained any serious psychological trauma. Thirdly, and finally, the operation was long and severe, and the patient frail and ill.

For our purposes the anaesthetic as a whole could only be described as excellent. Perfect surgical conditions were provided with the minimum risk to the patient.—I am, etc.,

CHARLES WELLS.
University of Liverpool. Professor of Surgery.

*** This correspondence is now closed.—ED., *B.M.J.*

Suture of Ruptured Kidney

SIR,—I should like to report a case of ruptured kidney which was successfully treated by suture.

The patient was a girl who attended the casualty department on November 16, 1946, following a fall from a bicycle. She complained of minor symptoms only, consisting of pain in the side, and was allowed to go home after her ribs had been strapped—following an *x*-ray examination which disclosed a fractured 11th right rib. However, she was brought by her mother again the same evening, as her condition had taken a very definite change for the worse, in that she had become pale and anxious and complained of increasing pain in the right loin and right side of her abdomen. She also had a brisk haematuria.

The diagnosis of ruptured kidney was then obvious, and she was admitted. Operation was performed the following day, and the right kidney was exposed through a loin incision. It was found to be ruptured by a transverse tear which extended into the pelvis, almost severing the kidney into two equal halves. There was a well-marked line of white tissue approximately 1 cm. in width on each cut edge. The kidney was approximated by three deep mattress sutures and, as it seemed viable and apart from the trauma quite healthy, was returned to the abdomen. The wound was closed with drainage, the tube being removed on November 24. The patient's subsequent progress was uneventful and she was discharged from hospital on December 4, 1946.

The case has been followed up until September 12, 1949, and has been examined by repeated intravenous pyelograms, which show good and equal function of both kidneys. The right occupies a slightly lower position, but appears to be quite healthy.

This operation was done without any previous experience of such a condition, and I imagine that such an injury is a relative rarity. However, I publish this case to show that such treatment can be successful and can apparently leave a kidney without any serious permanent damage.—I am, etc.,

Plymouth. R. D. N. BISSET.

Atomic Weapons and the Medical Profession

SIR,—The H bomb is now in process of construction, and Professor Einstein warns us that "annihilation of any life on earth has been brought within the range of technical possibilities." Until recently weapons of war were designed for the purpose of destroying military targets and killing combatants. Now, however, we have a new and outrageous situation. The new bomb has been described by American physicists as "no longer a weapon of war but a means of extermination of whole populations." Should this greatest threat to human life evoke no reaction from the medical profession? Our sole *raison d'être* as doctors is the helping and saving of life, and it is surely our duty to do what we can to prevent disaster from overtaking the human race. Doctors all over the world unite against the menace of bacteria. Surely they should unite against the menace of this "super pathogen."

I suggest that the leaders of the medical profession issue a solemn manifesto condemning the use of atomic weapons and, in the name of humanity, urging their abolition. They should press their colleagues in America and Russia to do likewise. Furthermore, we can influence people individually by stating our views and it is noteworthy that even the greatest people in every land have their own medical advisers. The medical

profession is an influential group in the world, and it could do much to rouse public opinion to that level at which it becomes an effective factor in history.

To some this letter may seem ineffectual verbiage, but surely it is preventive medicine of a peculiarly urgent nature.—I am, etc.,

Hove, Sussex. W. V. CRUDEN.

Students' Literary Style

SIR,—Mr. Ranger M. Whelan (February 18, p. 441) rightly associates the modern student's lack of literary style with the crowded and specialized curriculum, so different from the broad avenues of learning with which he has become acquainted in the higher forms at school. Equally to be blamed, however, is the teaching ineptitude of the university staffs in the faculty of medicine. During my own career as a medical student the good teachers could be counted on the fingers, and I believe the position is no better to-day.

Professors and lecturers in medicine are appointed on their academic and professional attainments, and no consideration is given to their teaching ability or inability whatsoever. Admittedly their qualifications should be of the first importance, but so also should be their ability to share their knowledge or to impart a thirst for it, especially in subjects so rigidly factual as medicine and surgery.

Occasionally the brilliant academician or clinician is naturally endowed with the gift of teaching, and such men become traditions in their schools, to be remembered with respect and gratitude by generations of students. But for the most part all who aspire to the lecture theatre should have a short course at a training centre for teachers if an improvement in the literary standard of medical students is desired.—I am, etc.,

Strichen, Aberdeen. WILLIAM HARKINS.

POINTS FROM LETTERS

Chilblains

Dr. ERNEST CULPIN (Southport, Queensland) writes: Dr. Henry S. Russell (December 24, 1949, p. 1473) asks, "Has anyone ever been cured of chilblains by taking calcium?" The answer is, yes, often. Belonging to a family with an allergic diathesis, I have been subject to chilblains for many years, even when living in North Queensland, and calcium has been an unfailing remedy. When serving in France during the 1914–18 war as an R.M.O. and at times with a field ambulance I was very liable to chilblains, but I always carried some calcium preparation and was able to keep the condition under control. The most satisfactory preparation for this purpose was, and still is, calcium chloride in 15-gr. (1-g.) doses. As it is a nasty preparation to take, it is dispensed with 2 dr. (7 ml.) of syrup, and chloroform water added to 2 oz. (57 ml.) for a dose, taken three times a day after food. I have frequently given this prescription to friends for the complaint and have never known it to fail. It is a surprise to me that the psychologist has not claimed that chilblains are a neurosis or something of the sort. There would probably be something in such a claim, for chilblains mostly occur in individuals with an allergic diathesis who are very prone to develop various minor psychoses.

Influenza Epidemics and General Practice

Dr. J. SACKVILLE MARTIN (Bognor Regis) writes: I wonder if you could spare me a few lines for reminiscence? Columns of statistics do not as a rule evoke memories, but those in Dr. W. J. Martin's paper on the death rate per million from influenza (February 4, p. 267) do so for me. One notes that from 1876, two years after I was born, to 1889 the disease was practically unknown. Then in 1890 it began, and in the two subsequent years the death rate rose to over 500 per million. I was then hovering between my last year of school and my first at Edinburgh University, and I remember my father rushing (if one rushes in a cab) all day, and also the puzzlement of the doctors who were faced with a new disease. My second reminiscence is coming back from the Army in 1917 to allow a colleague to have his turn, when I had two panels, 5,400 in all. Then the big epidemics of 1918 swept down on us. The figures of deaths are given as 3,129 per million for 1918 and 1,217 for 1919. Need I say more of the nightmare of those years, when one worked from morning until night and then knew that all one could do was entirely inadequate?

Obituary

J. A. RYLE, M.D., F.R.C.P.

Nuffield Professor of Social Medicine, Oxford

Professor J. A. Ryle, whose death in his sixty-first year was announced briefly in last week's *Journal*, was one of the most distinguished figures in contemporary medicine. After making his mark as an outstanding clinician and teacher at Guy's Hospital he was at the early age of 46 appointed Regius Professor of Physic at Cambridge. Searching no doubt for a way in which to put into practice those high ideals which he sought, he accepted in 1943 the appointment of Nuffield Professor of Social Medicine at Oxford. As the guest of honour at the Centenary Celebration of the New York Academy of Medicine in March, 1947, he defined social pathology "as the medicine and pathology of families, groups, societies, or larger populations. Just as human pathology is the related science of clinical medicine so may social pathology be viewed as the related science of social medicine." He considered that medical science and practice had evolved along individualist lines, but that now "the scientific study of health and disease in man . . . must henceforward concern itself to an ever-increasing degree with the interactions and correlations of disease and health with changing social circumstances." Professor Ryle stood for Cambridge University in 1940, and in his election address he stressed under-nutrition as a widespread cause of ill-health and put as a prominent feature in his programme "insistence on the proper maintenance of the social services and a fairer distribution of the burdens of war as between the well-to-do and poorer classes."

John Alfred Ryle was born in 1889, the son of Dr. R. J. Ryle, of Brighton. He was educated at Brighton College and Guy's Hospital, where he was awarded the gold medal in medicine. He qualified in 1913, served in the war of 1914–18, and in 1919 proceeded M.D. London with gold medal. In 1919 he took the M.R.C.P., and he was elected F.R.C.P. in 1924. He was the Goulstonian lecturer of the Royal College of Physicians in 1925 and Croonian lecturer in 1939, and Hunterian professor of the Royal College of Surgeons in 1932. From 1932–6 he was physician to His Majesty's Household, and from 1936 been Physician Extraordinary to the King. Ryle served on the B.M.A. Planning Commission and also on the committee which drew up the Charter for Health published in 1946. He was a member of the Medical Research Council from 1935 to 1939. During the recent war he acted as consultant adviser in medicine to the Minister of Health in the Emergency Medical Service. Before the war Ryle gave much time to the organization of Spanish relief and to work on behalf of refugee medical men and students, and from 1941 onwards he devoted much of his time and energy to helping forward the work of the Anglo-Soviet Medical Committee which was set up in September, 1941.

Professor L. J. Witts writes: By the death of Professor J. A. Ryle the medical profession has lost a leader who lived close to the Hippocratic ideal. Once, when he was travelling down the Wye in a canoe, a boy looking over a bridge shouted, "Here comes Gandhi!" The remark was truer than he knew. Ryle had a transparent honesty and serenity, but he had a vein of asceticism and a resentment of social injustice which grew in strength through the years.

John Alfred Ryle was born at Brighton in 1889, the third of a family of ten children. His paternal grandfather had been Bishop of Liverpool. His father was a doctor who had become a rationalist during his medical training. Ryle himself was a rationalist, and his faith was expressed in the little book *Fears May Be Liars* which he wrote during the second world war. Ryle went to Guy's, like his father, and by the time the first war started he had carried out with Izod Bennett the studies on the fractional test meal which made his name famous. He served from 1914 to 1919 and quickly showed the ability in recognizing and describing clinical syndromes which was one of his great gifts. His subsequent career fell into three phases. The first was the Guy's period, from 1919 to 1935, during which Ryle consolidated his reputation as a clinical scientist and consultant. Tall, fair, and handsome, he was popular with doctors for his diagnostic skill and with patients for the reassurance of his voice and presence. He was an able lecturer, and the fruits of this period of research and practice were collected in his book on the *Natural History of Disease*. He had intense respect for the value of the individual, and emphasized the study of the patient as a whole. He was a keen eugenist, much interested in the interplay of heredity and environment. He deprecated undue reliance on laboratory investigations, and advised restraint in any form of human experiment, whether in new treatments or in clinical scientific study. When he was being driven to hospital or to a consultation, he sat in the back of his car and read, nearly always from one of the old clinicians such as Sydenham or Hilton and rarely from a current journal. His conservatism was sometimes excessive, particularly as his impressive and persuasive manner endowed even his less considered judgments with weight. When the Americans introduced the liver treatment of pernicious anaemia he exclaimed, "What will they be doing next!"

Ryle's career seemed firmly charted for the positions of senior physician to Guy's, leading London consultant, and President of the Royal College of Physicians. To the general surprise he turned aside and accepted the Regius Chair of Physic in Cambridge in 1935. For some time he had been becoming more socialist and pacifist in outlook, and it may be that the life of a successful consultant no longer gave him the satisfaction without which its ardours are insupportable. For a while, however, Ryle seemed to have lost his way, and these middle years were his least productive period. It was partly that the set-up of the clinical school at Cambridge was unsuitable, for any system whereby a distinguished professor teaches or researches on colleagues' beds on a permissive basis is unlikely to work well. Moreover, Ryle was not a clubbable man and he disliked college life and entertainments. Cambridge between the wars had a large enough left wing to have enfolded him happily, but it was never his way to take the smooth with the rough or to realize that there are occasions on which it is meet to make merry and be glad even though the Yangtze is overflowing its banks and children are starving in Spain. How different from his predecessor, Langdon-Brown, who, when the Germans irrupted into the Low Countries, said, "Come, let's go and have a good dinner, it may be the last we shall eat!" Shortly before the war Ryle stood unsuccessfully for Parliament, where his talents would surely have been mis-spent. When war came again in 1939 he walked his garden in an agony of suspense before deciding that resistance to Fascism was more important than strict pacifism. He then drove himself hard in the struggle, both in East Anglia and back at Guy's in the Blitz, and in 1942 he had his first coronary thrombosis.

In 1943 he was invited to the Chair of Social Medicine at Oxford, and entered on a lustrum of great happiness and productivity. He had at last found the destination for which he had so long been searching. He knew that his expectation of life was diminished, and frankly regarded himself as a St. John the Baptist, preparing the way for the gospel of social medicine. He built a successful department and wrote a series of lectures on social medicine which were published in 1948 under the title *Changing Disciplines*. This book was well received. In it Ryle gave precision to his ideas of social medicine as a practice and as a scientific discipline; it will be for the future to decide how long these two horses can run in harness together. In spite of ill-health he travelled widely, to India, America, and South Africa. Many visitors came to see him at Oxford, and his fame was now greater than ever. A minor hypertensive attack in 1948 did not deter him, but in the summer of 1949 he had a severe coronary thrombosis from which he never recovered. He met his illness with remarkable tranquillity and continued to write poetry, which was one of his less familiar avocations. He was, indeed, a radiant spirit who showed what a luminous quality scientific humanism can possess, and his death is a personal bereavement to very many people, the loss of one to whom they turned when they were fretted by the cares of this world. He leaves a wife and five children to whom he was greatly devoted. He had the satisfaction of seeing two of his children qualify in medicine and two in science, while the fifth is nursing.

Mr. W. T. Russell, D.Sc., writes: As a very intimate colleague of Professor Ryle I would like to record my appreciation of him as the man I really knew—others can speak or write of him as the great clinician of international fame. Our association extended to nearly seven years, and my daily contact only helped to increase my affection for him. He had all the qualities which make for real greatness. There was, allied to a first-class brain, unfailing courtesy, sympathy, kindness, and tolerance. His magnetic personality was an inspiration, and every member of his staff loved him. His loss to the Institute and to Social Medicine is irreparable, but he will live in the memory of each one of us who had the privilege of being associated with him in the work in which he so strongly believed. As a measure of that belief I will quote from one of his last letters to me: "Notwithstanding all my happy years in clinical teaching and practice, this Institute chapter has been for me one of the best and most stimulating of all."

G. N. W. writes: At Guy's Hospital in 1929 I had the great good fortune to become house-physician to Dr. John Ryle, as he then was, an appointment which led to a friendship which ripened as the years passed. Others better qualified than I am will doubtless tell of his attainments in the professorial chairs at Cambridge and at Oxford, but I would like to speak of his influence on an ordinary practitioner. On his ward rounds or in his lectures he scorned the dogmatic or dramatic method, and my impression is that as a result he was not sufficiently appreciated

by the junior students, but he was the perfect teacher of the more advanced or qualified man. These who were able to take advantage of his balanced and humanitarian approach to the whole art of medicine must surely have been influenced for the rest of their professional lives. His gentle rebuke to an over-enthusiastic searcher after knowledge—"Don't you think it would be kinder to let this poor man die in peace even if we lose a little information?"—was typical of him. His understanding of the natural history of disease and the expected effects of various treatments was profound, and no one trained under John Ryle would be led astray by the latest blandishments of pamphlet or blotter.

It was always a stimulating experience to discuss any aspect of medicine with him, but his interests certainly did not end with professional matters, and to those who knew him well he was a most delightful talker and philosopher on all subjects. At the height of his powers, with much work uncompleted and several plans for research in social medicine on hand, the onset of crippling cardiac illness must have been a bitter blow, but he faced it with a serenity and courage which was indeed an example to those who knew him in his later days. Modern medicine would be the better for more like him.

Dr. BERNARD EDWARD GRANVILLE BAILEY died at his home in Midhurst on February 4 at the age of 76, after a long illness. A student at St. Bartholomew's Hospital, he qualified in 1899 and settled in Midhurst in 1902 after holding house appointments at the East London Children's Hospital and at the City of London Hospital for Diseases of the Chest. He was also a senior assistant medical officer at Shoreditch Infirmary. All of his life in general practice was spent in and around Midhurst. He loved the country and country people, and it was not only for his professional work and skill that he was loved in return, but also for his willingness to help anyone, rich or poor, when they were in trouble. He was a keen supporter of many activities of village life, and it is largely due to his energy and initiative that Midhurst has one of the best-equipped cottage hospitals in the county. He will be missed, not only by his colleagues, but by a wide circle of friends in this part of Sussex.—R. W. L.

Dr. RICHARD HENRY WAGNER died suddenly at his home in Plymouth on February 5, in his eighty-sixth year. Graduating at the Queen's University, Cork, in 1886, he settled in Plymouth, where he continued in practice for some fifty years. He enjoyed remarkably good health and remained alert until the end of his long and useful life. Throughout his career Dr. Wagner was keenly interested in everything that affected the interests of the profession. His sound judgment, prudence, and lucidity made him the ideal representative in committee or conference. He held many offices. He was chairman of the Plymouth Division of the B.M.A. in 1923–5, president of the South-Western Branch in 1926–7, and president of the Plymouth Medical Society, of which he was made the first honorary life member. He served as chairman of the Prince of Wales's Hospital, and he was also chairman of the local insurance committee. Through his death Plymouth has lost one of its oldest and most highly esteemed doctors. Ever cheerful, he was gifted with an abundance of wit and humour. His many friends and colleagues will extend their sincere sympathy to his widow and daughter.—G. D.

Dr. ROBERT D'ERNST, of Geneva, died on February 7. The death of this public-spirited doctor will be regretted not only by his professional colleagues but also by the many doctors' families who benefited through the Swiss organization, the A.S.E.M., over which he presided. This body provided a channel through which the Swiss profession was able to offer hospitality to the orphaned sons and daughters of medical men in other countries. It will be recalled that in 1947 some 70 British children were enabled, through the good offices of Dr. d'Ernst, to spend a three months' holiday in Switzerland. Dr. d'Ernst was a chevalier of the French Legion of Honour and of the Order of Leopold of Belgium.

Dr. LILIAS FRAZER-NASH died on February 8 at the age of 90. She was among the first women to qualify in medicine in this country, studying at the London School of Medicine for Women, and taking the Scottish triple qualification in 1887. Her first medical appointment after qualifying was in Lahore, where her husband was in the Indian Civil Service. They were married there soon after her arrival, and shortly afterwards Dr. Frazer-Nash took over the duties of medical superintendent at Hyderabad Hospital. She was there for three years, working with the most primitive equipment, but attending to the needs of 80 to 100 patients a day. Her eldest son, Captain A. Frazer-Nash, the well-known racing motorist and aircraft engineer, was born at Hyderabad, and three years later, after the death of her husband, she returned to England, where her second son was born. At this stage Dr. Frazer-Nash went into private practice, became medical officer to the Salvation Army homes at Hackney, and then went on to specialize in the treatment of mental cases in private homes at Hampstead and Hendon. When she retired from practice she settled in Kingston, and in 1927 was elected to the borough council. She gave valuable service on the education and public health committees, and she was also for many years a member of St. Paul's Church Council. During the war Dr. Frazer-Nash was bombed out of her home in Kingston Hill, and she spent the last few years of her life at her son's home. There are many who will remember the good work she did in India in the early days, and the sense of public spirit which inspired all her activities at Kingston.

Dr. HAROLD SHELDON died with tragic suddenness on February 18 at his home in Southport. He had just returned from the reunion dinner of a school to which he had been medical adviser for 25 years. Harold Sheldon graduated M.B., Ch.B. at Owens College, Manchester, in 1912, proceeding M.D. in 1920. After resident appointments at the Manchester Royal Infirmary and at Warrington he joined the R.A.M.C. He landed in France on August 16, 1914, and took part in the retreat from Mons. Later in the war he came to Southport to take charge of an auxiliary military hospital. On being demobilized in 1919 he joined the late Dr. Baildon in general practice, and when Dr. Baildon died he continued the practice single-handed. In 1937 Dr. Sheldon was president of the Southport Medical Society, and in 1938–9 he was chairman of the Southport Division of the B.M.A. He served the St. John Ambulance Association well as examiner, corps surgeon, and later chairman of the local association. For many years he was deputy obstetrician to the Christiana Hartley Maternity Hospital. A gifted musician, Dr. Sheldon had written hymns, chants, and songs, though he published only a few of them. The recessional played at his funeral was his own composition and was inspired by Kipling's well-known verses. Dr. Sheldon endeared himself to his patients. He was devoted to them and to his work, keeping abreast of progress, and being always willing and eager to avail himself of the help of his colleagues or to help them. Many patients came to him for advice and assistance in matters other than those immediately concerned with health. His acts of kindness were many but unobtrusive. Dr. Sheldon leaves a widow, a daughter, and two sons, of whom one has begun a career in radiology. To all his family the sympathy of his colleagues and friends will be extended.—W. A. M.

Surgeon Commander J. V. Smith writes: I was very sorry to read of the death of Dr. L. G. BOURDILLON (January 28, p. 255). I met him only once, but his kindness and courtesy made a very great impression on me when, as a Naval medical officer wandering eastwards with the unaccustomed charge of a tent hospital unit, I took advantage of a delay at Port Said to glean some hints from military authorities in the Suez Canal area. Someone put me in touch with the A.D.M.S. (Colonel Bourdillon), who not only drove to fetch me and spent half a day showing me hospitals and camps under canvas, but throughout that time made me feel a welcome guest rather than the

extraneous nuisance that I must have been. Such an attitude is encouraging to individual strangers and promotes friendship between the medical services, to the ultimate benefit of their patients.

Medical Notes in Parliament

The PRIME MINISTER, Mr. Clement Attlee, announced the names of the members of the new Cabinet on February 28. Mr. Aneurin Bevan continues as Minister of Health and Lord Addison as Lord Privy Seal.

Dr. Edith Summerskill, who was previously Parliamentary Secretary at the Ministry of Food, is now the Minister of National Insurance in place of Mr. James Griffiths. Mr. Maurice Webb takes over from Mr. John Strachey as Minister of Food.

The new House of Commons met for the first time on Wednesday, March 1, and Colonel Clifton Brown was again chosen as Speaker. On the same day in the House of Lords Lord Haden-Guest was introduced, took the oath, and subscribed the Roll. Lord Haden-Guest, who was formerly Dr. Haden Guest, was sponsored by Lord Webb-Johnson and Lord Shepherd.

At the opening of the first session of the new Parliament on Monday, March 6, the King, in his Address, referred to the improvement of water supplies, particularly in rural areas, and to the introduction of legislation to effect this as soon as circumstances permitted.

In view of the restricted time available and the heavy volume of financial business to be transacted, the Government proposed only a limited programme of legislation for the present session. Nevertheless, should other measures prove immediately necessary for the maintenance of full employment and the national well-being they would be submitted to Parliament, even though they might prove contentious.

A Bill would be laid before the House to amend the Medical Acts so as to raise the standard of medical education and to modify the constitution and disciplinary procedure of the General Medical Council.

Legislation would also be introduced to vary the constitution of the Central Midwives Board and to make other alterations in the law relating to midwives.

Medico-Legal

"T 35" FOR TUBERCULOSIS

Recently at the West London Court, Albert Otto Dobbs, a chef, of Gloucester Road, Kensington, was fined a total of £10 for unlawfully advertising by circular and letter a preparation known as "T 35." In this case, which was brought under the Pharmacy and Medicines Act of 1941, the prosecution was undertaken by the Pharmaceutical Society of Great Britain. Apart from the unlawful advertising, Dobbs also pleaded guilty to selling "T 35" in a package which did not carry a label showing its composition.[1]

Mr. A. C. Castle, for the Pharmaceutical Society, said that in reply to a letter sent by an inspector of the society asking for particulars of his treatment for tuberculosis, Dobbs replied that "T 35" had been used as a cure for tuberculosis in Germany for 27 years. The substance had been discovered by a Bavarian woman, and was grown in the southern parts of Europe. The quantity required for a course of treatment cost £5. There was a stipulation that the treatment must be superintended by a doctor.

A cheque for £5 was sent, and the "T 35" was duly received. It appeared to consist of ground laurel berries, a well-known herb, the cost of which was approximately 2s. a pound. Dobbs stated that he was only an agent in this country for the preparation, and said that he did not think he was committing an offence because he did not put an advertisement in the newspapers.

[1] *West London and Fulham Gazette*, Sept. 9, 1949.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

Dr. R. E. M. Pilcher has been appointed a governor of Boston Grammar School by the Council of the Senate.

UNIVERSITY OF GLASGOW

On February 25 the degree of M.D. was conferred, *in absentia*, on Oliver Gray.

UNIVERSITY OF LONDON

At a meeting of Convocation on January 21 Desmond Kyran Mulvany, M.S., F.R.C.S., and Napier Arnold Thorne, M.D., M.R.C.P., were elected to the Standing Committee of Convocation for a period of three years (*Corrected announcement*).

UNIVERSITY OF SHEFFIELD

Lord Boyd-Orr, F.R.S., will deliver the fourth Basil Hicks Lecture in the University on Friday, November 10. The title of the lecture will be announced later.

The Basil Hicks Lecture was ounded by the ate Dr. William Hicks (Principa of Firth College and University College, Sheffield, and Charter Vice-Chancellor of the University) in memory of his son who was killed in the war of 1914–18. The foundation provides for a triennial lecture, each lecture to deal with some aspect of the Great War, either in its origins, its conduct, or its social and international consequences, or such other subject of national or international interest as may be arranged. Previous Basil Hicks Lecturers have been Mr. H. A. L. Fisher, Dr. Gilbert Murray, and General Smuts. The lecture was in abeyance during the second World War.

The following appointments have been made: *Assistant Lecturer in Physiology*, E. W. Clarke, M.B., Ch.B.; *Assistant Lecturer in Pathology*, F. N. Ghadially, M.D.; *Honorary Lecturer in Industrial Medicine*, R. A. Trevethick, M.B., Ch.B.

Dr. A. H. Morris has resigned from the post of Demonstrator in Anatomy in the University.

ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH

At a quarterly meeting of the College held on February 7, with the president, Sir David K. Henderson, in the chair, the following were elected to the Fellowship: Drs. Alexander Brown, R. A. K. Harper, and R. Brockie Hunter.

The following candidates were elected to the Membership: Drs. E. R. Gauld, I. R. Gray, W. T. Strauss, E. S. Thodey, J. A. C. Wilson, J. Ram, W. P. Small, N. R. Grist, L. G. Leitch, W. R. M. Alexander, J. B. Wilson, P. F. Bartley, W. J. H. Lord, D. B. Nathan, T. Joyce, A. A. B. Swan, K. J. Keeley, Jessie E. J. Grainger, and C. H. M. Walker.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW

A Loyal Address has been received by the Secretary of State for Scotland from the Fellows of the Royal Faculty of Physicians and Surgeons of Glasgow for presentation to the King on the occasion of the 350th anniversary of the foundation of the Faculty and has been presented accordingly.

At a meeting of the Royal Faculty of Physicians and Surgeons of Glasgow on February 6, with the President, Dr. W. R. Snodgrass, in the chair, S. I. A. Laidlaw was admitted Fellow of Faculty *qua* physician and John Hewitt Fellow of Faculty *qua* surgeon.

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

The first John Shields Fairbairn Memorial Lecture will be delivered by Professor Arthur T. Hertig, professor of pathology at the Boston Lying-in Hospital and associate professor in the Department of Pathology, Harvard Medical School, at the College (58, Queen Anne Street, London, W.) on Tuesday, March 21, at 5 p.m. His subject is "Hydatidiform Mole and Chorio-epithelioma." Admission is by ticket only, obtainable from the secretary.

The Services

Lieutenant-General Sir Neil Cantlie, K.B.E., C.B., M.C., late R.A.M.C., has been appointed Honorary Surgeon to the King, in succession to Brigadier A. E. Richmond, C.B.E., retired.

Major-General Thomas Menzies, O.B.E., late R.A.M.C., has been appointed Honorary Physician to the King, in succession to Major-General J. C. A. Dowse, C.B., C.B.E., M.C., retired.

Brigadier A. L. Crockford, D.S.O., O.B.E., M.C., T.D., R.A.M.C., T.A., has been appointed Honorary Surgeon to the King in succession to Major-General Sir Ernest M. Cowell, K.B.E., C.B., D.S.O., T.D., retired.

EPIDEMIOLOGICAL NOTES

Population Estimates for 1949

On June 30, 1949, the population of this country was estimated to be 43,595,000, with 9,497,000 under 15 years of age. These figures are given by the Registrar-General in the second of a new series of publications giving up-to-date estimates of the populations of each local government area in England and Wales.

These estimates differ from those previously issued in that they include the members of the armed Forces stationed in each area; previous estimates related to civilians only.

Discussion of Table

In *England and Wales* there were increases in the notifications of measles 275, scarlet fever 233, and dysentery 19, while the largest decrease was in the incidence of acute pneumonia 39.

There was a small increase in the incidence of scarlet fever in many areas; the largest rises were Lancashire 69 and Glamorganshire 43. Changes in the trend of measles occurred in only a few areas; the largest rises in incidence were Kent 108, Suffolk 80, and Surrey 60, while the largest falls were Norfolk 89 and Durham 79. Except for an increase of 40 in Yorkshire West Riding, the changes in the notifications of whooping-cough were small. Increases in the notifications of diphtheria were reported from Lancashire 8 and Durham 5; these two counties together accounted for half the total notifications of diphtheria.

Only 1 case of dysentery was notified in Nottinghamshire, Nottingham C.B., where 20 cases were recorded in the preceding week. The notifications of dysentery in Yorkshire West Riding rose from 78 to 140 (Leeds C.B. 41, Bradford C.B. 36, Sheffield C.B. 27, Dewsbury C.B. 12, and Shipley U.D. 15). In Lancashire the notifications fell from 103 to 50 (Stretford M.B. 22, Manchester C.B. 10). The other large centres of dysentery were Durham 33 (Sedgefield R.D. 9, Gateshead C.B. 6, and South Shields C.B. 6); London 22 (these were scattered throughout the metropolis and not more than 4 cases were reported in any borough); Glamorganshire 19 (Cardiff C.B. 18); Gloucestershire 18 (Bristol C.B. 18); and Northumberland 18 (Newcastle-upon-Tyne C.B. 17). Of the 10 cases of typhoid fever 5 were notified in London.

In *Scotland* there were increases in the notifications of dysentery 55, scarlet fever 45, and whooping-cough 33. A fresh outbreak of dysentery with 28 cases was notified in Perth. In Dundee the incidence of dysentery increased from 13 to 25; in Glasgow from 12 to 23; and in Edinburgh from 2 to 10. A rise in the incidence of scarlet fever occurred throughout the country.

In *Eire* there were rises in the notifications of measles 61 and whooping-cough 54, while the incidence of scarlet fever decreased by 31. The rise in the notifications of measles was contributed by Carlow, Ennis R.D. with 64 notifications, and the decline in the incidence of scarlet fever by Dublin C.B. Two fresh outbreaks of whooping-cough were reported in Westmeath, Delvin R.D. 50, and in Meath, Dunshaughlin R.D. 20.

In *Northern Ireland* a decrease of 13 in the notifications of whooping-cough was the largest change in the totals of notifications, although the totals for the separate counties showed fairly large fluctuations. In Belfast C.B. there were increases in the incidence of measles 33 and scarlet fever 28.

Week Ending February 25

Notifications of infectious diseases in England and Wales during the week included: scarlet fever 2,064, whooping-cough 1,679, diphtheria 74, measles 4,980, acute pneumonia 1,098, acute poliomyelitis 38, dysentery 445, paratyphoid fever 6, typhoid fever 10. In the great towns 78 deaths were attributed to influenza.

No. 7

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended February 18.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1950					1949 (Corresponding Week)				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Meningococcal infection	59	6	17	—	—	30	2	17	3	1
Deaths		1	1				—	—		
Diphtheria	80	8	21	2	3	148	16	32	8	4
Deaths	1	—	—	—	—	2	—	—	—	—
Dysentery	344	22	97	—	—	68	16	19	—	2
Deaths					—				—	—
Encephalitis, acute	3	—	—	—	—	—	—	2	—	1
Deaths		—		—			—			
Erysipelas			31	12	4			29	14	6
Deaths										
Infective enteritis or diarrhoea under 2 years				13					27	
Deaths	25		3	6	—	43		10	3	—
Measles*	4,526	71	77	137	347	18,441	809	111	123	144
Deaths†			—	—	—				1	—
Ophthalmia neonatorum	24	2	8	—	—	41	6	12	—	—
Deaths										
Paratyphoid fever	6	—	1 (B)	—	—	5	—	—	—	—
Deaths	—	—	—		—	—	—	—		—
Pneumonia, influenzal	1,189	78	64	—	13	1,139	78	46	18	21
Deaths (from influenza)‡	76	8	18	3	—	94	11	24	—	—
Pneumonia, primary			363	¶				391	48	
Deaths	353	49		16	28	394	98		14	11
Poliomyelitis, acute	39	3	—	5	1	18	2	1	1	—
Deaths§	9	1		—		1	—			
Puerperal fever		—	4				—	4		
Deaths										
Puerperal pyrexia‖	80	9	6	—	3	93	4	6	—	2
Deaths							—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	2,130	134	233	89	148	1,417	104	268	121	37
Deaths†			—	—	—			—	—	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Typhoid fever	10	5	4	1	—	7	—	—	—	—
Deaths	—	—	—	—	—	—	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Whooping-cough*	1,652	68	401	103	52	3,011	212	239	87	71
Deaths	5	—	1	—	—	10	—	1	1	1
Deaths (0–1 year)	331	44	48	32	13	341	39	47	27	22
Deaths (excluding stillbirths)	6,249	953	864	258	172	6,926	1247	852	239	163
Annual death rate (per 1,000 persons living)			17·4					17·1	14·8	
Live births	7,720	1223	941	507	210	7,612	1264	936	403	249
Annual rate per 1,000 persons living			18·9					18·8	25·0	
Stillbirths	215	23	36			203	26	32		
Rate per 1,000 total births (including stillborn)			37					33		

* Measles and whooping-cough are not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales and Eire.

¶ Primary pneumonia no longer notifiable in Eire.

Medical News

Nurses' Memorial Fund

Lord Moran, P.R.C.P., will broadcast at 8.45 p.m. on Sunday, March 12, in "The Week's Good Cause" in aid of the British Commonwealth and Empire Nurses War Memorial Fund, of which he is chairman of appeals. The fund was launched in 1946 to provide a worthy war memorial to the nurses and midwives of the British Commonwealth and Empire, and now stands at over £71,000. It is hoped to reach £250,000 to form a fund to establish postgraduate travelling scholarships for nurses. The furnishing of a war memorial chapel in Westminster Abbey, which forms the second object of the fund, has been provided for by the gifts of the nursing profession. At least £16,000 is needed as a capital sum to found each scholarship of not less than £350 per annum, although some donors, including the Royal College of Physicians, are giving seven-year scholarships under deed of covenant. An interim award of scholarships given outright in this way is being made this year, and candidates are being selected from the United Kingdom, Canada, Pakistan, Ceylon, Hong Kong and Singapore, and Malaya. Gifts to the British Commonwealth and Empire Nurses War Memorial Fund can be sent to Lord Moran, Chairman of Appeals, Appeals Office, 12, Whitehall, London, S.W.1.

Lectures on the Common Cold

Dr. C. H. Andrewes, F.R.S., director of the research unit working on the common cold at Salisbury, is lecturing in Italy for the British Council on "Influenza" and "The Common Cold" between March 8 and 22.

Joint Tuberculosis Council

Dr. Norman England, of Oxford, recently resigned the honorary secretaryship of the Joint Tuberculosis Council. His successor is Dr. R. L. Midgley, Hawkmoor Sanatorium, Bovey Tracey, South Devon (Telephone: Bovey Tracey 2107). All communications should be addressed to Dr. Midgley. The other officers of the council are unchanged: chairman, Dr. Peter W. Edwards; vice-chairmen, Professor F. R. G. Heaf and Dr. N. Tattersall; honorary treasurer, Dr. A. P. Ford.

Peckham Health Centre Closing

The pioneer health centre at Peckham is to close through lack of funds. Dependent upon voluntary contributions, it costs about £25,000 a year to maintain. The experiment was started in 1926 by Dr. G. Scott Williamson and Dr. Innes H. Pearse, and the present building was erected in 1935. After being closed temporarily during the war the centre was reopened after it, but has since been in financial straits. Negotiations are in progress for its development as a National Health Service experimental health centre, and a plan has been submitted to the L.C.C.

Midland Institute of Otology

At the annual general meeting of the institute, which was founded in 1947 for the encouragement of otolaryngological research, the following were elected as officers:—President: Mr. W. Stirk Adams; vice-president: Mr. W. L. Thomas; honorary secretary. Mr. Reginald S. Strang; honorary treasurer: Mr. W. H. Lomas; members of council: Mr. B. S. Carter, Mr. J. B. Cavenagh, Mr. E. Collins, Mr. Robert Evans, Mr. F. Brayshaw Gilhespey, Mr. Hamilton Baynes, Mr. Sydney Vernon.

Pharmaceutical Society

The Privy Council has nominated Dr. J. A. Giles, who was Chief Inspector under the Vivisection Acts, and Dr. B. A. Young, of St. Alfege's Hospital, Greenwich, to be members of the council of the Pharmaceutical Society of Great Britain.

Colour-televised Dentistry

A demonstration of colour television for dental instruction was a feature of the recent annual clinical meeting at Guy's Hospital Dental School. Guy's Hospital has already a permanent black-and-white television installation for teaching in the surgical department, but this colour installation was put in temporarily by the makers—the Pye organization—at the request of the dental school. In the course of the day Professor W. E. Herbert, Professor M. A. Rushton, and Mr. A. R. F. Thompson showed cases and performed operations which were watched by groups of about 50 people on television screens in another part of the building. The operating-chair was surrounded by three banks of fluorescent lights, and transmission was by closed circuit, operated on the same 405 lines per frame as the existing B.B.C. service.

Prize for Research in Leukaemia

The Robert Roesler de Villiers Foundation, Inc., of 417 Park Avenue, New York, 22, is offering a prize of $500 (or in special circumstances $1,000) for the most significant contribution to the knowledge of the nature, causes, origin, treatment, and cure of acute leukaemia and allied conditions, either published or accepted for publication by a reputable journal in or outside the United States of America, between January 1, 1950, and October 20, 1951. In exceptional circumstances the publication requirement may be waived. The prize has been instituted in memory of Robert Roesler de Villiers, who died of acute leukaemia at the age of 16½ years on October 20, 1944. Further particulars may be obtained from the Foundation itself, from Dr. Sven Moeschlin, Kantonsspital, Zurich, or from Dr. S. Haberman, 3301, Junius Street, Dallas, Texas.

Licence to Practise in U.S.A.

In the last fifteen years more than 10,000 medical practitioners trained outside the U.S.A. have migrated to that country. Some State boards exclude all foreign medical graduates from taking their licensing examinations, while others admit them to the examinations. The American Medical Association is carrying out an inquiry into the standards of medical education in foreign universities, and in a preliminary report it has announced (*J. Amer. med. Ass.*, 1950, **142**, 587) that graduates of all the universities in the United Kingdom may be considered on the same basis as graduates of approved medical schools in the United States. The approval is not extended to licentiates of the Royal Colleges or Scottish Corporations, nor does it absolve graduates from having to take licensing examinations in the U.S.A.

Goitre in Ceylon

Dr. Dagmar Wilson, of Oxford, has left Geneva for Ceylon, where as a WHO expert consultant she will undertake an investigation on the incidence of endemic goitre. This survey will be carried out under the auspices of the World Health Organization at the request of the Ceylon Government. Dr. Wilson was a member of the Women's Medical Service of India from 1916 to 1925, and has represented the Government of India at many international conferences on child welfare. She held a nutritional research appointment in India under the Royal Society (1937–40), and since 1943 has been attached to the University Laboratory of Human Nutrition, Oxford.

Cancer Co-ordinating Committee

The Liverpool Regional Board announces that the following members have been appointed to the Cancer Co-ordinating Committee under the National Health Service:—Six members of the Regional Hospital Board: Professor Sir Henry Cohen, Professor W. M. Frazer, Dr. D. Brown, Mr. T. Keeling, Alderman D. Plinston, and Alderman N. Birch. Four members of the board of governors: Mr. A. N. Denaro, Professor E. A. Owen, Mr. A. A. Gemmell, and Mr. P. H. Whitaker. Two members of the University: Professor C. A. Wells and Mr. Duncan Norman. Two members of the Radium Institute Management Committee: Mr. H. T. Wilson and Dr. J. MacAllister. Two additional members will be appointed at the first meeting of the committee.

Assistant Director-General of WHO

Major-General Sir Sahib Singh Sokhey, of India, has been appointed Assistant Director-General of the World Health Organization in succession to Dr. Raymond Gautier. He was director of the Haffkine Institute, Bombay, specializing in vaccine and serum production, and is an authority on bubonic plague and cholera.

Fluoride for Dental Caries

The Ministry of Education has announced that over 3,000 school-children in various parts of England will shortly take part in a test of the effectiveness of water containing fluoride as a protection against dental decay. There are only a few areas in this country where the water contains fluoride, but there are good grounds for believing that a comparable effect is produced by wetting the teeth on several occasions with a solution containing fluoride. Because of the shortage of dentists it is not possible to offer the treatment at present to all children in the country, but it will be made available for a limited number. It will be given by the staffs of a number of dental hospitals and of local education authorities. The participating bodies are: the Eastman Dental Clinic, London (in co-operation with the London County Council); Newcastle Dental Hospital; Newcastle Local Education Authority; Leeds Local Education Authority; West Riding Local Education Authority; Sheffield Dental Hospital; Sheffield Local Education Authority; Birmingham Dental Hospital; Bristol Local Education Authority; and possibly Middlesex Local Education Authority. The methods of selecting the children may vary according to local arrangements, but no child will receive the treatment without the parents' consent. Both boys and girls will be included, but it is not proposed at present to give the treatment to any child over 12 years, since such children would have left school before the full effects of the treatment could be observed.

University Grants Committee

Professor G. W. Pickering, whose term of office on the University Grants Committee expired on November 1, 1949, has consented to serve for five more years. He is professor of medicine in London University and director of the Medical Unit at St. Mary's Hospital.

COMING EVENTS

British Medical Students Association

The following arrangements have been made for the week March 10 to 17. *London Hospital.*—March 14 (5.30 p.m.): Lecture by Dr. Alice Stewart, Assistant Director of the Institute of Social Medicine, Oxford, on "Social Medicine." *St. Andrews University Medical Society.*—March 10: B.M.A. Lecture by Dr. W. N. Pickles.

Biological Assays

A joint meeting of the Biometric Society (British Region) and the Biological Methods Group of the Society of Public Analysts and Other Analytical Chemists will be held at 2.45 p.m. on Tuesday, March 14, at the Medical Society of London, Chandos Street, Cavendish Square, London, W.1. There will be a symposium on "The Design and Evaluation of Biological Assays." The symposium will be preceded by the annual general meeting of the Biometric Society at 2 p.m.

Forensic Medicine Congress

The Third Congress of the International Academy of Forensic and Social Medicine has been postponed. It was to have been held at Cairo on March 21–26.

Chronic Rheumatic Diseases

A week-end course on "The Chronic Rheumatic Diseases," with lectures, ward rounds, and practical demonstrations, will be held at the Rheumatism Unit of St. Stephen's Hospital on Saturday and Sunday, March 25 and 26. The course will be opened by Professor C. S. Pickering, whose subject will be "The Significance of the Effect of Compound E in Rheumatoid Arthritis." Sir Adolphe Abrahams will preside.

Registrars' Group: Scottish Western Region

A general meeting of all the registrars employed within the Western Region of Scotland will be held in the Lister Theatre, Glasgow Royal Infirmary, on March 22, at 7.30 p.m.

Medical Art

An exhibition of medical art by members of the Medical Artists Association of Great Britain will be held by the Royal Society of Medicine from March 27 to April 1. Admission is free.

Birmingham and Midland Obstetric and Gynaecological Society

Professor A. T. Hertig, professor of pathology at Harvard University, at the Boston Lying-in Hospital, and at the Free Hospital for Women, Boston, Mass., will deliver a lecture, under the auspices of the Birmingham and Midland Obstetric and Gynaecological Society, on "Observations on the Genesis of Cervical and Endometrial Carcinoma with respect to the *in situ* Stage" in the Medical School, Birmingham, on Wednesday, March 29, at 4.30 p.m. The lecture is open to all graduates and undergraduates.

North of England Ophthalmological Society

Dr. Ramón Castroviejo, of New York, will deliver two lectures before the North of England Ophthalmological Society. The first will be given in the Clinical Theatre, Royal Infirmary, Manchester, on Thursday, March 30, at 3.30 p.m., when his subject will be "Cataract Surgery—Usual and Unusual Procedures." The second lecture will be given at the Medical School, Leeds, on Friday, March 31, at 3 p.m., and the title of the lecture will be "Keratoplasties and Keratectomies." Both lectures will be illustrated by films and slides. All ophthalmologists are invited, and those wishing to attend one or both lectures should communicate immediately with Mr. W. M. Muirhead, D.O.M.S., honorary secretary, 70, Upper Hanover Street, Sheffield, 3.

SOCIETIES AND LECTURES

Monday

LONDON UNIVERSITY.—At University College (Eugenics Theatre), Gower Street, W.C., March 13, 5.30 p.m., "*Statistical Problems Connected with Medical Diagnosis*," by Professor Jerzy Neyman (University of California).

MEDICAL SOCIETY OF LONDON, 11, Chandos Street, Cavendish Square, W.—March 13, 8.30 p.m., "*Peripheral Arterial Disease*," discussion to be introduced by Professor A. M. Boyd and Dr. Geoffrey Evans.

UNIVERSITY COLLEGE, Gower Street, London, W.C.—March 13, 5.30 p.m., "*The Non-specificity of Nervous Conduction*," by Professor B. Rensch (University of Münster).

Tuesday

BRITISH MEDICAL STUDENTS ASSOCIATION: LONDON HOSPITAL BRANCH.—At London Hospital Medical College, Turner Street, Whitechapel, E., March 14, 5.30 p.m., "*Social Medicine*," by Dr. Alice Stewart.

CHADWICK PUBLIC LECTURE.—At Westminster Medical School, 17, Horseferry Road, London, S.W., March 14, 2.30 p.m., "*The Maintenance of Health by the Prevention of Accidents in the Home*," by Dr. C. A. Boucher (Ministry of Health).

CHELSEA CLINICAL SOCIETY.—At South Kensington Hotel, 47, Queens Gate Terrace, London, S.W., March 14, 7 for 7.30 p.m., discussion: "*Modern Intra-thoracic Surgery*," to be opened by Mr. R. C. Brock and Dr. Paul Wood.

INSTITUTE OF DERMATOLOGY, 5, Lisle Street, Leicester Square, London, W.C.—March 14, 5 p.m., "*Fungous Diseases of the Skin*," by Dr. I. Muende.

INSTITUTE OF OPHTHALMOLOGY (UNIVERSITY OF LONDON), Judd Street, London, W.C.—March 14, 5.30 p.m., "*Amaurosis Fugax et Amaurosis Fulminans*," by Dr. S. Behrman.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., March 14, (1) 3.30 p.m., *X*-rays and museum specimens by Mr. J. E. Semple; (2) 5 p.m., "*Survey of Different Methods of Treatment of Prostatic Enlargement*," by Mr. R. Ogier Ward.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, S.W.—March 14, 5 p.m., "*Investigation of Thyroid Function and Disease by the Use of Radioactive Iodine*," Oliver-Sharpey Lecture by Dr. E. E. Pochin.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—March 14, 5 p.m., "*Villous Tumours of the Rectum*," Hunterian Lecture by Professor M. R. Ewing.

Wednesday

GLASGOW UNIVERSITY: DEPARTMENT OF OPHTHALMOLOGY.—March 15, 8 p.m., *"Glioma of the Retina,"* by Dr. J. D. Fraser.

INSTITUTE OF DERMATOLOGY, 5, Lisle Street, Leicester Square, London, W.C.—March 15, 5 p.m., *"X-ray Technique,"* by Dr. C. W. McKenny.

INSTITUTE OF PSYCHIATRY (UNIVERSITY OF LONDON), Maudsley Hospital, Denmark Hill, London, S.E.—March 15, 4.30 p.m., lecture-demonstration for postgraduates by Dr. E. Stengel.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., March 15, 5 p.m., *"Malignant Disease of the Prostate,"* by Mr. J. D. Fergusson.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland Place, London, W.—March 15, 3.30 p.m., *"The Provision and Maintenance of a Safe Water Supply for the Consumer,"* by Lieutenant-Colonel E. F. W. Mackenzie, M.B., Ch.B., D.P.H.

ROYAL MEDICAL SOCIETY, 7, Melbourne Place, Edinburgh.—March 15, 7 p.m., annual extraordinary general meeting.

ROYAL MICROSCOPICAL SOCIETY.—At B.M.A. House, Tavistock Square, London, W.C., March 15, 5.30 p.m., ordinary meeting. *"Golgi Apparatus and Mitochondria and Their Significance in Cellular Physiology,"* by Dr. G. H. Bourne.

Thursday

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck Street, London, W.—March 16, 8.15 p.m., *"Megavoltage X-rays,"* papers by Drs. H. Miller, M.A., Ph.D., and J. H. Martin, Ph.D., A.Inst.P.

EDINBURGH CLINICAL CLUB.—At B.M.A. Rooms, 7, Drumsheugh Gardens, Edinburgh, March 16, 8 p.m., (1) *"The Rhesus Factor in Midwifery,"* by Dr. E. C. Fahmy; (2) business meeting.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., March 16, (1) 3.30 p.m., ward round by Mr. H. G. Hanley; (2) 5 p.m., *"Transvesical Operations on the Prostate,"* by Mr. A. R. C. Higham.

LONDON ASSOCIATION OF THE MEDICAL WOMEN'S FEDERATION.—At B.M.A. House, Tavistock Square, London, W.C., March 16, 8.30 p.m., (1) Extraordinary general meeting; (2) *"Recent Work of the Institute of Social Medicine, Oxford, on the Epidemiology of Pulmonary Tuberculosis,"* by Dr. Alice Stewart.

NATIONAL HOSPITAL, Queen Square, London, W.C.—March 16, 5 p.m., *"The Cerebral Forms of Disseminated Sclerosis,"* by Dr. J. Haguenau (Paris).

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—March 16, 5 p.m., *"Investigation of Thyroid Function and Disease by the Use of Radioactive Iodine,"* Oliver-Sharpey Lecture by Dr. E. E. Pochin.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—March 16, 5 p.m., *"Respiratory Responses to Operative Trauma,"* Hunterian Lecture by Professor E. M. Nanson.

ROYAL SOCIETY, Burlington House, London, W.—March 16, 11 a.m., *"The Measurement of Growth and Form,"* discussion to be opened by Professor S. Zuckerman, F.R.S.; 4.15 p.m., Election of Fellows.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—At Royal Army Medical College, Millbank, London, S.W., March 16, 7.30 p.m., laboratory meeting. Various demonstrations will be given.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—March 16, 4.30 p.m., *"Neurology,"* lecture-demonstration by Dr. Denis Williams.

UNIVERSITY COLLEGE HOSPITAL MEDICAL SCHOOL, University Street, Gower Street, London, W.C.—March 16, 4.30 p.m., *"The Experimental Approach to the Problem of Diabetes Mellitus,"* Sydney Ringer Memorial Lecture by Professor F. G. Young, F.R.S. (University of Cambridge).

Friday

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck Street, London, W.—March 17, (1) 5 p.m., meeting of medical members; (2) 6.30 p.m. to 8 p.m., Symposium: *"The Biological Effects of Non-ionizing Radiations with Particular Reference to Short Waves and Micro Waves,"* papers by Dr. A. C. Boyle and Messrs. W. E. Willshaw, M.Sc., A.M.I.E.E., H. F. Cook, B.Sc., F.Inst.P., H. Burton, B.Eng., and T. S. England, B.Sc., F.Inst.P.

FACULTY OF RADIOLOGISTS.—At Royal College of Surgeons of England, Lincoln's Inn Fields, London, W.C., March 17, 2.15 p.m., Therapy Section Meeting. Discussion: *"Carcinoma of the Bladder,"* to be opened by Mr. D. S. Poole-Wilson, Dr. J. M. W. Gibson, and Dr. J. L. E. Millen.

INSTITUTE OF OPHTHALMOLOGY (UNIVERSITY OF LONDON), Judd Street, London, W.C.—March 17, 5.30 p.m., *"The Central Connexions of the Visual Pathways,"* by Dr. F. Stansfield.

MAIDA VALE HOSPITAL FOR NERVOUS DISEASES, Maida Vale, London, W.—At Medical School, March 17, 5 p.m., neurological demonstration by Dr. P. H. Sandifer.

SOCIETY OF PUBLIC ANALYSTS.—At Royal Society, Burlington House, Piccadilly, London, W., March 17, 4.30 p.m., annual general meeting, followed by the Bernard Dyer Memorial Lecture by Sir John Russell, F.R.S.

WEST LONDON MEDICO-CHIRURGICAL SOCIETY.—At South Kensington Hotel, 41a, Queen's Gate Terrace, London, S.W., March 17, 7.15 for 7.30 p.m., dinner meeting. *"Atomic Energy,"* by Sir George Thompson, F.R.S.

Saturday

MIDDLESEX COUNTY MEDICAL SOCIETY.—At Shenley Hospital, near St. Albans, Hertfordshire, March 18, 3 p.m., general meeting.

YORK MEDICAL SOCIETY.—At 17, Stonegate, York, March 18, 8.30 p.m., *"Hormones,"* by Professor Margaret Fairlie.

APPOINTMENTS

Dr. Theodore Farnworth Anderson, O.B.E., has been appointed a Nominated Official Member of the Legislative Council of the Colony of Kenya.

CUNNINGHAM, ROBERT C., M.B., Ch.B., D.P.H., D.P.M., Senior Medical Officer in the Mental Health Section of the Health Department of the County Council of Essex.

EAST ANGLIAN REGIONAL HOSPITAL BOARD.—*Consultant Chest Physician in Great Yarmouth and Lowestoft Area*, I. M. Young, M.B., Ch.B.; *Consultant Chest Physician in Huntingdon and Isle of Ely Area*, C. E. Downes, M.R.C.P.; *Consultant Psychiatrist and Deputy Medical Superintendent Little Plumstead Colony*, J. J. Ryan, M.B., B.Ch., D.P.H., D.P.M.; *Consultant Anaesthetist in Peterborough Area*, H. J. Richardson, L.R.C.P.&S.Ed., L.R.F.P.S.Glas., D.A.; *Medical Registrar, White Lodge Hospital, Newmarket*, W. F. Rogers, M.D.

FARQUHAR, ROBERT W., M.B., Ch.B., D.P.H., Divisional Medical Officer, Health Division No. 13, Lancashire County Council.

HASTILOW, IRENE M. D. F. C., M.B., Ch.B., D.P.H., D.C.H., D.Obst.R.C.O.G., Assistant County Medical Officer, Kent County Council (except the District of Bexley).

HILL, L. C., M.D., F.R.C.P., Director of the School of Physiotherapy, Bath.

LILWALL, BERNARD G. A., D.S.O., M.S., F.R.C.S., Consultant Surgeon, Canterbury and Isle of Thanet Groups of Hospitals.

LIVERPOOL REGIONAL HOSPITAL BOARD.—*Consultant Non-resident Deputy Physician-Superintendent at Newsham General Hospital (formerly Belmont Road Hospital)*, W. Fine, M.D., M.R.C.P.; *Consultant Orthopaedic Surgeon to St. Helens Hospital*, A. G. O'Malley, M.Ch.Orth., F.R.C.S.Ed.

MORRISON, JOHN BLACK, B.Sc., M.D., Medical Superintendent, Abergele Sanatorium, North Wales.

ROBINSON, CHARLES B., M.D., F.R.C.P.I., D.P.M., Resident Medical Superintendent, Purdysburn Mental Hospital, Saintfield Road, Belfast.

ROSS, THOMAS, M.B., Ch.B., D.P.H., Medical Officer of Health, County Borough of Walsall.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Brewerton.—On February 5, 1950, at Stanboroughs Maternity Home, Watford, to Sylv'a (née Sturridge), wife of Dr. R. Brewerton, a son.

Kipling.—On February 10, 1950, at the Townend Maternity Home, Hull, to Joan Kipling (née Hollins), B.M., B.Ch., wife of John Kipling, M.A., D.Phil., a daughter.

Wyman.—On February 26, 1950, at Westminster Hospital, to Joan (née Beighton), wife of John B. Wyman, M.B.E., D.A., a son.

DEATHS

Brown.—On March 3, 1950, at Towyn, Merioneth, Eric Barlow Brown, M.B., Ch.B., late of Union Street and Chamber Road, Oldham.

Charsley.—On February 27, 1950, Robert Stephen Charsley, M.R.C.S., L.R.C.P., J.P., formerly of Slough, Bucks.

Duigan.—On March 2, 1950, Victor John Duigan, M.R.C.S., L.R.C.P., of Gorleston, late of East Dereham, Norfolk, aged 73.

Flewitt.—On February 26, 1950, at 48, Abbey Lodge, London, N.W., Charles York Flewitt, M.B., B.Ch., aged 73.

Fuchs.—On February 20, 1950, Hans Karl Fuchs, M.D., of 19, Liskeard Gardens, Blackheath, London, S.E.

Gallagher.—On February 21, 1950, at Hillingdon Hospital, Uxbridge, Middlesex, Edward James Masterson Gallagher, M.B., Ch.B.

Gibbs.—On February 26, 1950, at "Whins," Hook Heath, Woking, Andrew Armstrong Gibbs, L.R.C.P.&S.I., & L.M., Major-General, I.M.S., retired.

Gibson.—On March 2, 1950, at Firs End, Heath Road, Petersfield, Percy Lempriere Gibson, M.R.C.S., L.R.C.P., Surgeon Captain, R.N., retired.

Graham.—On February 19, 1950, Matthew Joseph Graham, M.B., B.Ch., D.P.H., late Malayan Medical Service, of 10, Clarinda Park West, Kingstown, Eire

Harker.—On March 2, 1950, William Edmund Harker, O.B.E., M.D., Surgeon Captain, R.N.V.R., of Newcastle-upon-Tyne, aged 79.

Harvey.—On February 23, 1950, after an operation, William John Saundry Harvey, D.S.O., M.R.C.S., L.R.C.P., Lieutenant-Colonel, R.A.M.C., retired, aged 79.

Hodge.—On March 2, 1950, at an infirmary, Glasgow, Ronald Dingwall Hodge, M.B., Ch.B., of Ardoch, Bianefield, Stirlingshire.

Keay.—On March 1, 1950, at Dunfermline and West Fife Hospital, Andrew Campbell Keay, M.B., Ch.B., late of Rosskeen, Kelty, Fife.

Laslett.—On February 22, 1950, at 21, Bolton Road, Farnworth, Lancs., Thomas George Laslett, M.R.C.S., L.R.C.P., J.P., aged 90.

Milne.—On December 25, 1949, John Milne, M.B., Ch.B., of West Hill, Sunderland Co. Durham.

Robinson.—On February 21, 1950, at Aldersyde, Dringhouses, York, Francis Aidan Robinson, M.C., M.B., B.S., D.P.H., Major, R.A.M.C., retired, aged 64.

Ryle.—On February 27, 1950, at his home in Sussex, John Alfred Ryle, M.D., F.R.C.P., aged 60.

Sheldon.—On February 18, 1950, at 14, Albert Road, Southport, Lancs., Harold Sheldon M.D.

Watt.—On February 23, 1950, at a nursing-home in Edinburgh, Robert Honey Watt, M.D., of Langholm, Dumfriesshire.

White.—On February 21, 1950, at 61, Hamilton Street, Saltcoats, Ayrshire, John Allan White, M.B., Ch.B.

Whitnall.—On February 19, 1950, Samuel Ernest Whitnall, M.D., F.R.S.C., of 324, Woodstock Road, Oxford.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Frogmen, Firemen, and Carbon Dioxide

Q.—*I read with interest the answer (October 29, 1949, p. 1000) to a question on the symptoms experienced when breathing through a closed-circuit apparatus, and Dr. Rollason's observations on the same point (December 24, 1949, p. 1484). Both refer to the reactions experienced when the apparatus is working satisfactorily and presumably the carbon dioxide is quite low. What are the sensations with a faulty absorption of CO_2, with an increasingly high percentage of CO_2 breathed in an atmosphere of excess oxygen, such as might occur with a self-contained diving apparatus of "frogman" type? To what level can the CO_2 rise before it becomes dangerous, and what sensations would warn the user of the apparatus that the absorption system was beginning to function inefficiently? The Siebe-Gorman breathing apparatus for fire-fighting, for instance, incorporates a soda-lime canister. How long is this likely to remain effective for absorbing exhaled CO_2 when a considerable amount of muscular exercise is undertaken?*

A.—The signs and symptoms of rising percentages of carbon dioxide in a closed circuit containing excess oxygen have not been adequately studied. As the inquirer suggests, the problem was one of the greatest importance during the war for underwater warfare. In experiments that have not yet been published it was found that if a subject is at rest under these conditions he will appreciate an accumulation of carbon dioxide due to a faulty canister. He will become aware of increased breathing due to the stimulation of the respiratory centre. He will also notice a sensation of warmth and flushing due to the considerable peripheral vasodilatation. However, before the carbon dioxide reaches dangerous levels he will reject the apparatus as he finds the overbreathing intolerable. In contrast to this, when the subject is exercising and already ventilating considerably it is possible to pass the stimulating range of carbon dioxide and enter the anaesthetic range without untoward discomfort. A number of "frogmen" in training were lost before this fact was appreciated. Those who survived stated that they felt as if they were "going under gas." In a series of experiments involving exercising and re-breathing into a Douglas bag containing oxygen, up to 13.8% of carbon dioxide was reached before the subjects became aware of acute symptoms. The symptoms encountered were variable. All the subjects were flushed and perspiring and a number showed dilated pupils. Tremors, usually of the arm, and a characteristic "jitteriness" were seen. Towards the end, some of the subjects had twitching of the arms, legs, or face. If they continued they became extremely dissociated, uncoordinated, and finally fell. Unconciousness was encountered on only a few occasions, and the subjects recovered in a few seconds. Transient vertigo and prolonged severe headache occurred on resumption of air breathing.

Generalizing, symptoms that may endanger a wearer of apparatus were encountered when the percentage of carbon dioxide was as low as 6%. Warning symptoms were of a vague nature, and, as with anoxia, the subject appears unable to appreciate abnormalities which are obvious to an observer. It is therefore imperative that there must be no doubt concerning efficient carbon dioxide absorption when breathing oxygen. Sir Leonard Hill described, many years ago, how subjects who filled their lungs with oxygen and exercised, while holding their breath, became ataxic and confused while continuing to exercise; it is likely that the same phenomenon was involved in these more recent experiments. A small proportion of the subjects find the stimulating effects of carbon dioxide intolerable even while exercising on oxygen, and such persons will avoid the higher and more dangerous percentages of carbon dioxide.

With fire-fighting and similar apparatus the period of safety with different types of canisters depends on the activities and the oxygen consumption of the wearer. Accurate figures can be given by the manufacturers. However, it should be the practice of all those responsible for the lives of men wearing self-contained oxygen breathing apparatus to avoid passing the stated half-life of any canister. This is particularly important when men are exercised under dangerous conditions. Any absorbent that is not absolutely fresh, even though it has not been used, should be rejected. This is not because of any lack of confidence in the keeping properties of the absorbents under proper conditions, but unless supervision is meticulous there is a grave risk of previous use or deterioration. Lives should not be risked to save a few shillings.

Teething Troubles

Q.—*In treating the teething problems which cause disturbed nights for both parents and the infant I have used chloral hydrate, but even in doses in excess of those usually recommended it seems to have little effect. What is the most satisfactory drug for this purpose for infants aged 6–12 months?*

A.—The pain of dentition is not easily relieved by the type of sedative which can be safely used for small children. Fortunately chloral is well tolerated, and doses up to 5 gr. (0.32 g.) can be given for infants of the age mentioned. Bromides are sometimes added, but there is little to commend this. Phenobarbitone in doses of ¼ gr. (8 mg.) or as soluble phenobarbitone is a useful alternative. Local applications to the gums sometimes help, especially the rubbing in of a little menthol in paraffin (1 grain to the ounce of paraffin : 2 mg. per g.). It is worth noting that the restlessness of the infant may not necessarily be due to dentition. Indigestion, early pink disease, earache, and possibly rickets are all in the list of disorders which may arise in the first 6 to 12 months of life. Large doses of vitamin D will sometimes produce calm when chloral has failed.

Streptomycin in Eye Infections

Q.—*Has streptomycin been used locally in eye infections? If so, with what results and for what conditions is it especially indicated?*

A.—Indications for the use of streptomycin locally in ocular infections are still largely undetermined. For tuberculous conjunctivitis, and possibly tuberculous iritis, streptomycin would have to be used systemically. The chief indication for the local use of streptomycin is superficial infection with *B. pyocyaneus* or similar Gram-negative bacilli. Ointments containing 100 mg. per g. of ointment base appear to be effective. The ointment base is of some importance; a mixture of 90% petroleum jelly and 10% liquid paraffin into which streptomycin is incorporated without solution is best. The ointment should be applied at hourly or two-hourly intervals according to the severity of the infection. If the cornea is involved it is best to use subconjunctival injections. Streptomycin of the purest type should be used, 0.5 g. being dissolved in a solution consisting of 10 min. (0.6 ml.) of sterile water and 5 min. (0.3 ml.) of adrenaline 1 : 1,000. Injections should be given every 24 hours. Generally it is not possible to give more than three or four injections owing to the local reaction. Most infections of the cornea by Gram-negative bacilli are, however, brought under control by this procedure, and subsequently treatment with streptomycin can be maintained by means of the ointment.

Infective Hepatitis

Q.—*What is the latest treatment for infective hepatitis? How long should the patient, a boy of 14 in this case, be away from school or work, and how soon after returning may he resume games? Should a liver function test be performed before any patient is allowed to resume full activities?*

A.—The essential points in the treatment of infective hepatitis are rest in bed and a diet containing liberal quantities of protein and carbohydrate. In at least 80% of cases the disease is so mild and recovery so rapid that a rigid dietary regime is unnecessary. Where the condition is more prolonged and more

severe, a daily allowance of 120 g. of protein and 300–400 g. of carbohydrate is desirable; the quantity of fat must depend on the patient's tolerance. Supplements of vitamins, particularly those of the B group, should be given. The patient should remain in bed until bilirubin has been absent from the urine for three consecutive days, and should do no more than sit in his room for a few hours daily while urobilinuria persists. Convalescence is slow, and vigorous exercise should not be permitted for at least a month after a mild attack. The disease is so variable that hard and fast rules cannot be made, but there can be no question that strenuous exertion can precipitate a relapse if undertaken too early. A gradual return to full activity should be planned, and restrictions reimposed if there is pain over the liver, undue tiredness, or dyspepsia. If the patient has reached full activity in this manner without any recurrence of symptoms, and if the liver and spleen have returned to normal, a liver function test is unnecessary. In most instances the patient's symptoms are a more accurate guide to his progress.

Passive Immunity Against Rubella

Q.—*Does convalescent human serum convey passive immunity against rubella? Assuming the danger of homologous serum jaundice can be eliminated, is there justification for injecting such serum into women immediately they are found to be pregnant, if they have never previously contracted the disease? In the absence of a more specific prophylactic, such inoculations, repeated if necessary, might tide them over the danger period in early pregnancy.*

A.—There is no convincing evidence that convalescent human serum or gamma globulin prepared from normal serum gives protection to those exposed to the risk of rubella. If a protective serum were available it would certainly be desirable to give an injection to any woman in the earliest stages of pregnancy who was known to be exposed to the infection. It would obviously be impracticable to give the serum to all pregnant women.

Action of "Priscol"

Q.—*Have vasodilators derived from imidazoline, such as "priscol," any long-term toxic effects—for example, on the bone marrow? What is the mode of action? What is the usual dosage, and can it be continued indefinitely?*

A.—The toxic effects of such drugs are on the whole immediate rather than long-term. They consist of flushing, tachycardia, tingling, nausea, and digestive upsets. Postural hypotension may occur, and "priscol" should not be given to patients with coronary disease, which not infrequently co-exists with some of the peripheral vascular disorders for which the drug is used. "Priscol" also stimulates gastric secretion, and should be used with caution in patients who have a history of peptic ulceration. It does not affect the bone marrow adversely. The vasodilator action of such drugs is not brought about by direct local action, but is due to their sympatholytic or anti-adrenaline effect. Obviously, only arterial spasm can be affected beneficially, and not organic occlusion. The optimal dose varies from patient to patient, and each case must be considered as an individual problem. The drug is given orally, and it is wise to start with a small dose, say a 25-mg. tablet three times a day after meals, which can be gradually increased, according to the reaction of the patient, to a maximum of 50 mg. four times a day. Administration can be continued for long periods, and in Raynaud's disease it is recommended that the drug should be given continuously throughout the winter.

White Children in the Tropics

Q.—*Is there any evidence of physical, intellectual, or moral impairment in white children brought up in a tropical climate? If this is so, and my experience in the Belgian Congo suggests that it is the case, at what age should such children be sent home in order to avoid these ill effects? Is there any difference in this respect between boys and girls?*

A.—There may be evidence of physical, intellectual, or moral impairment in white children brought up in a tropical climate, but there appears little sound evidence that these effects are due to the climate alone. Certain parasitic infections are commoner in tropical climates, and psychological influences may be very different from those a white child experiences when brought up, say, in this country. The effects of such factors often seem to be confused with the effects of the climate alone. European children often grow too rapidly if kept in tropical climates above the age of 10 or 12 years. They may also lack the companionship of children of their own age, and they tend to be spoilt by coloured servants; thus they may easily become domineering and bad mixers.

NOTES AND COMMENTS

Anemone Contact Dermatitis.—Dr. L. B. BOURNE (London, N.) writes: With reference to the question and answer on this subject ("Any Questions?" February 18, p. 448) I would suggest, from experience in industry where sensitizers to many chemicals is a problem, that a barrier cream which will protect specifically against the juices of anemone is the best answer to the problem. I have been permitted to examine the records of a barrier cream manufacturer, and it would appear that the risk from primula, etc., can be completely overcome by a barrier cream. From an investigation carried out by the agricultural research station of a leading drug company a report has been received as follows: "Results of testing sample creams X and Y as a preventive of primula plant-rash on hands, wrists, and forearms were entirely satisfactory in both cases. Deliberate application of the under sides of leaves of several vigorous plants—applications lasting five minutes—to the areas mentioned produced no rash or irritation, even on the soft underarms. Normally the slightest contact produces virulent poisoning." A report has also been received from a county agricultural executive committee: "However, I can say that so far samples A and B have been used in connexion with tomatoes both in a dry and moist state, and most samples have proved extremely satisfactory. Cleansing of the hands has been reduced to a minimum without any subsequent roughening of the skin. Sample C has so far been used in connexion with the handling of chrysanthemums, and, while it was impossible to obtain a worker susceptible to dermatitis, the results are promising, as cleansing has been reduced to a minimum." In view of the fact that agricultural workers after being exposed to the hazards may be far from good washing facilities, and as a result the skin is in contact with the sensitizing agent for longer periods than in industry, it appears to me that a barrier cream is the only answer to the problem, but it must be pointed out that the barrier cream must be specific against the particular sensitizing agent.

Holidays Abroad.—Colonel A. V. O'BRIEN (late I.M.S.) writes from the Albergo San Remo, Via d'Azeglio 36, Rome: Having been in Italy for over a year during the war and resident in Rome and elsewhere for the past year, I take the liberty of disputing the reply given in "Any Questions?" about holidays abroad (February 11, p. 385). While reasonable care should be exercised in drinking water, it is a gross exaggeration to say that "unboiled water should be forbidden except in large towns." As for the antimalarial precautions suggested, they are simply ludicrous. The Army position in Italy in 1946 was quite different. I wonder what precautions the writer would suggest against the possibility of the cross-Channel steamer sinking or the railway carriage going on fire. These are at least as likely as the contraction of malaria on the way to Rome. Malaria is for all practical purposes non-existent north of Rome. Therefore to subject the Boy Scouts to such a regime is nothing less than unnecessary torture. I would also point out that medical reciprocity has *not* been restored since it lapsed at the entry of Italy into the war. The drugs suggested are all readily available in even the small villages of Italy and at a less cost than in the U.K. There is no point in exporting a small chemist's shop. If the questioner would like any further information I shall be only too pleased if he will communicate with me.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 2111. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

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